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A CLASSIFICATION OF TEETH THE DISEASED PULPS AND APICES OF WHICH ARE RELATED TO INFECTIVE FOCAL AND SYSTEMIC SEQUELAE[1]

CLARENCE J. GRIEVES

Baltimore, Maryland

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I. INTRODUCTION

So heated is current discussion of so-called "pulpless," "dead," or "non-vital" teeth; so many bodily ills of doubtful etiology, hence treated experimentally, are said to have been caused by "non-vital" teeth and cured by the removal of such teeth from the maxillae; so doubtful pathologically are these ills; so questionable is the right to the claim "cured," when applied either to the chronic *systemic* or to the equally chronic *oral* diseases; that these and other reasons prompt a pause by both dental and medical professions, in order the better to classify and define the pathology, not only of "pulpless" teeth, but of all other teeth, invaded by pathogenic bacteria.

[1] Paper read before the First District Dental Society of the State of New York, at a meeting in New York City, February 2, 1920. See page xxxv of the Section on Proceedings of Dental and Stomatological Societies for a report of a discussion of this paper at that meeting by Drs. W. B. Dunning, Charles F. Ash, N. T. Shields, and M. L. Rhein, of New York City; and Dr. Grieves.

Such a working classification (16), crude but practical, was presented by the writer eighteen months ago; and, recently, at the last meeting of the National Dental Association. It will be attempted again, with your forbearance, this evening; but it must be accepted as a clinical presentation only, for, as the matter now stands, it is next to impossible for dentists to consult intelligently among themselves upon this subject. How, then, can they expect their medical confrères to comprehend in oral pathology what, to their own minds, is not clear? Is it any wonder that our medical confrères, not understanding, condemn all "filled pulpless teeth," overlooking many others more virulently diseased, and doubt the judgment of any dentist who honestly insists that certain types of diseased teeth are both harmless and functional, hence should be retained?

Any intelligent discussion of this subject is necessarily based upon certain facts, which must be considered axiomatic, even though subject to correction in the light of the constantly accruing results of research, like all other scientific data. Upon such clinical axioms we must all agree, just as we accept and apply treatment to that disease entity known as dental caries, while disagreeing as to its etiology. These facts should be couched in the language common to general, not merely to oral, pathology, so that all pathologists may understand when we speak of the etiology and pathology of pulpal and apical diseases, all of which, if properly classified, would indicate the correct treatment and prognosis. Not only would this course greatly clarify the subject in our own minds and save countless, otherwise functional, teeth, but it would promote a better understanding, and closer coöperation, between both dental and medical professions.

If many of the facts submitted are bromidic and appear too elementary, let us remember that eventually we must return again and again to first principles; it is only by orientation of these, to recent methods of practice, that real truth will survive. Your attention is, therefore, directed briefly to the histopathology of the two main dental portals by which pathogenic organisms approach the pulp and subapical tissues, and thereby the lymph channels and blood stream, namely, (a) the dentin, pulp and root apex, and (b) the gingival crevice.

(II. Section II has been put out of sequence, on pages 330 and 331, in order to present the whole of the classification on opposite pages.)

III. DISCUSSION OF THE CLASSIFICATION

Class I.—Average teeth; pulps vital

Class I, which may be carious, while both pulps and apices are vital and healthy, is necessarily a broad one, since all teeth that have been carious or restored by filling, within the prescribed limits of the class, belong to this group and lead to a moment's consideration of dental caries.

Many phases of tooth destruction exist which cannot be classed under erosion, abrasion, or caries. However, the disease group known under the broad term, dental caries, must be so accepted. Like many other wasting diseases, its etiology is unsettled. Whether we hold with those who consider acidophilic forms as the only cause; or believe these active in symbiosis with cocci; or again, that various streptococcal strains are the only factors, three facts are assured clinically:

(1) Streptococci constitute 50 per cent of all mouth growths of microörganisms and often are apparently normal in healthy oral and pharyngeal mucosae [Kligler and Gies (22); Hartzell and Henrici (19).]

(2) Pathogenic coccal forms have been demonstrated repeatedly, by culture and in slides, lying deep in the tubuli of vital dentin, under soft caries, and after thorough cavity preparation. [Miller (25), Goadby (9), Hopewell-Smith (21), Hartzell and Henrici (19), Rosenow (30).]

(3) Miller (25) and others identify cocci as the deepest invaders of the dental pulp.

Attention is called to this septic factor in all stages of dental caries, hence:

> *Conclusion 1.* Dental caries is dual in character, being a chronic tooth-wasting disease, with tissue necrosis or liquefaction superficially, and pathogenic forms irregularly invading the hard underlying dentinal matrix. This sepsis begins in the granular layer of Tomes, and is the primary infective focal portal. Therefore, because dentin is hard, or free from soft caries, it is not necessarily free from bacteria; and some effort must be made to sterilize every cavity-preparation in vital teeth.

II. INFECTIVE PORTALS OF PULPALLY AND APICALLY DISEASED TEETH CLINICALLY CLASSIFIED. (AGE AND USE ARE ALWAYS MODIFYING FACTORS)

Class I. Average teeth; pulps vital. Neither pulpal nor periapical symptoms.

May be carious or filled; coronal dentin infected, but pulps healthy; apices vital.

Class II. Sound teeth; pulps diseased by pyorrhea. Gingival and periodontal symptoms.

Pulps involved by lymphocytic infiltration and infection—48 per cent of cases; apical hyperplasia, occasionally, depending on gingival disease and stress (occlusal) or

Class III. Abraded or eroded teeth. Occasional pulpal symptoms.

Dentin: tubular calcification and hyalin; apical hyperplasia. Periapical disease may result from pulp exposure (bacterial), or degenerative pulp products (chemical), but is slow.

Pulps involved by adventitious dentin; calcific, fibroid, fatty, degeneration; pulp nodules; more rarely, necrosis or gangrene

Class IV. Average teeth; pulps diseased.

By trauma..... Pulpal thrombosis; infarct; or

By senility..... Apical hyperplasia; fibroid atrophy; or

By surgical exposure..... May be controlled, when not infected; or end in

Occasional pulpal or periodontal symptoms.

Infective pulpitides

Hyperemia: active (arterial)

Hyperemia: passive (venous)

Destructive

Acute, simple: May end in

Productive

Chronic hyperplastic

Acute, purulent: May end in

Fast—Odontoblasts and fibrils liquefied; complete dentinal and apical infection likely.

Chronic ulcerative

Slow — Odontoblasts and fibrils vital, sometime, particularly middle third and apical third

Class V. Carious teeth; pulps infected.

All pulpal, and a few periapical, symptoms.

Apices vital. Subgingival and apical dentin, and cementum, not usually infected.

Pulps involved by caries and degeneration, or both; subgingival pulp infected, via vessels, but products of degeneration (chemical) or of infection (bacterial) have not passed the foramina to produce periapical disease. If not properly treated and filled, or "inoperable" due to root deflection and multiple foramina, the teeth of Class V will surely drift into Class VI. If, by modern pulp-canal surgery, the apices are left vital, not infected, not over-medicated, not perforated, or not over-filled, this group is hopeful, and may be filled and retained as harmless and functional.

Class VI. Infected teeth; septic pulps.

Canals filled or unfilled

All periapical and no pulpal symptoms.

Periapical infections

Destructive: open, granulating; fistulous; exudative	Productive: closed; granulating
Parulis	Proliferating periodontitis
Acute alveolar abscess	Granulomata ending in ↓
Chronic alveolar abscess	Diffuse granulating areas or cysts
Granulation tissue; pus; sero-fibrinous exudate; polymorphs	Lymphocytes; plasma; cells; fibrous sac; condensing osteitis; sclerosis

Apex: necrotic and hypoplastic; subgingival and apical dentin and cementum septic.

Pulpal abscesses, purulence and gangrene, putrefactive and infective products of which pass beyond the foramina, producing the above named diseases; result also from too radical, or negligent, root-canal medication and surgery.

It is likely that periapical disease is produced by many acute stages of pulpitis, with little damage to the apex, which is left vital, or to the apical periodontium, that is not irreparable. Fistulous abscesses may so result, which may be cured; all other forms are incurable.

It is probable that such latent infection exists in every well filled tooth, as it does in every carious tooth. Fortunately, the infection is merely latent and rises to the dignity of disease only when it involves the pulp. Disease is prevented (a) orally, by scientific filling-restoration, checking recurrent caries; and (b) cavally, by dentinal tubular calcification, granular hyalin formation or adventitious dentin—the usual dentinal protection, of which Hopewell-Smith (21) says: "Probably nearly every dentinal change is accompanied by some healing process of the pulp." This resistance reverses the rule, being less active in young adolescent dentin, with many tubuli or little matrix substance, than in adult or middle life. If, by reason of rapid and virulent caries, or a lack of resistance, these pulp reactions break down, much chronic pulp disease may occur under good fillings, with solid but infected dentin intervening, eventually producing periapical disease, while the dentin is sensitive and the pulp vascular.

We should be mindful of this infective element in treating children's teeth, particularly first molars. A deep cement filling or pulp capping (the proper procedure) often fails to protect the pulp because of collateral dentinal infection. Possibly, these are the most insidious and dangerous of all periapical types of infection.

What is conservative practice for Class 1, in the light of the foregoing? With dentinal infection controlled, and the wonderful pulpal resistance still functioning in a vast majority of cases, do we stand with the "devitalists," who would remove a pulp before it becomes infected, because sometime this function may fail? Or, have we such confidence in our canal technic as to devitalize and positively (?) remove every particle of pulp, and fill every root, where occurrences of apical deflection and multiple foramina are at least 40 per cent (16), with perfect root fillings in only 7 per cent of the cases (of thousands) and with 47.5 per cent of all filled roots apically diseased?

The operator who proceeds to devitalization, in spite of these facts, assumes a reparative skill more potent than the human body possesses. If he succeeds, it is usually not by his skilful technic, important though that may be, but in spite of it—by remarkable reparative processes in the periodontium and vital apex. Hence:

Conclusion 2. The normal pulps of vital teeth should be devitalized rarely, and only when devitalization is important for the construction of such occlusal restorations as are impossible by other methods and absolutely necessary to general nutrition; recalling the 70 per cent of occlusal efficiency of bridges, as compared with 30 per cent in the case of dentures.

The possibilities of modern partial dentures and attachments, which have been so markedly improved, conserving rather than destroying attaching teeth, must be thoroughly studied and exhausted before it is decided to risk devitalization of a normal pulp.

Class II.—Sound teeth; pulps diseased by pyorrhea

It is not proposed, in discussing Class II, to dwell upon gingival diseases, except as they tend to produce pulpal or periapical disease.

Dewey and Noyes (7, 8) have demonstrated the association of three sets of lymph vessels with the blood supply that completely surrounds every tooth, which is seated, so to speak, in a basket of periodontal membrane, dense socket lining (lamina dura), and blood vessels, all of which tissues these lymph vessels penetrate, nourish, and drain. The largest group passes from the gingiva, centrally down through the membrane, paralleling the cementum; it is joined by gingival and middle-third groups, the first of which carries infection from the gingival crests to the nearest lymph nodes. Thus, a superficial gingival infection would be deflected over the interseptal crests; but a periodontal infection would finally reach the subapical tissues, splitting the periodontal membrane on its way, as in a true pyorrhea pocket.

At this point we would impress you with the results of recent research by Henrici and Hartzell (20) who, in studying one hundred coronally sound but pyorrheal teeth, found 48 per cent of the vital pulps invaded by lymphocytic infiltration and the vessel walls much damaged, with coccal forms definitely established in the tissue. These findings should settle the question of hematogenous pulp infection. The round-cell infiltration is a counterpart of the cell reaction in adjacent tissues, previously noted, by Latham (23, 24)

and Talbot (31), as pulp degeneration, associated with interstitial gingivitis. But the presence of pathogenic bacteria is a new highly significant fact, about which centres many possibilities, among them the following.

(1) Periapical disease is not coincident with gingival lesions; it does not arise in the same percentage of cases, nor occur in 48 per cent of sound pyorrheal teeth the pulps of which show lymphocytic invasion and pathogenic bacteria. On the contrary, casual clinical observation shows that many pulpless teeth, 47.5 per cent of which are apically diseased, are the only teeth remaining firm and free from gingival disease in an entire denture swaying in purulence. This may be due to sclerosis or a condensing osteitis, which often involve the apical- and middle-third attachments of such teeth, making them firmer than those adjacent. This condition, by partially destroying the lower lymph system, would present a barrier against further periodontal invasion.

(2) That this 48 per cent incidence of pulp pathology does not always produce a like amount of periapical disease may be explained by the fact that, coincident with pulp disease gingivally induced, necessarily runs a pyorrhea that destroys the gingival-third fibers, and creates a compensating hyperplasia of the apical-third fibers and cementum to meet extra stress. Thus, the accessory, often the main, foramina, are closed and the entire apex is encased with laminae of adventitious cementum, which may seal this portal against infection, either afferent or efferent. Further, in any serious loss of gingival tissue resulting in exposure of cementum, pulpal fibrotic and calcific degeneration is common. This interferes with complete pulp removal and root-canal filling, which, in these cases, is below the average for all pulpless teeth. In fact, the high percentage of failures to fill apices and the low percentage of periapical diseases is notable, apical hyperplasia (and often a real hypercementosis) commonly existing, having closed this whole region against both pulpal and gingival infection, before the dentist could reach it.

Be these hypotheses what they may, it is safer to assume, for teeth involved by gingival disease, that sepsis might at any time reach apical and pulp tissues, since the main lymph flow is from gingiva to subapical tissues; and to add, to the usual conservative rule (15),

that "pyorrheal teeth, gingivally damaged to the middle third and swaying in purulence, must be removed," the further practice stated in the following conclusion:

> *Conclusion 3.* Any pulpless tooth, the periodontal membrane of which is gingivally purulent, should be removed, no matter how perfectly filled, or how well repaired subapically, whether periapically diseased or not; for, at any time, it might become diseased by the gingival pathway. If pulp devitalization is absolutely necessary, it should be practised on only such pyorrheal teeth as are not swaying to the middle third; or those cured (?) by peridontic treatment; and then, on only those in which apical hyperplasia has closed in part the foramina as noted. (Such hyperplasia is usually demonstrable, clinically and by good radiographs.)

The fact that 48 per cent of the pulps of gingivally diseased teeth are infected will not, it is to be hoped, lead to a new fad for the destruction of these pulps—just as all pulpless teeth are now being removed because they are interstitially infected. The situations are analogous and comparable. It has been shown that real periapical disease (one of the good reasons for tooth extraction) does not result in anything like the same proportion, from demonstrated pulp disease, as it does from the best efforts to remove such pulps.

A large percentage of dentinal infections never reaches the pulp to produce pulpitis, just as, no doubt, a similar percentage of cases of pulpitis never reaches the subapical tissues to induce disease outside the tooth. These are latent infections constantly occurring elsewhere throughout the body, in connective tissue, particularly bone, from the vascularity of which they are definitely walled off by sclerosis and repair. You are referred, in this connection, to the remarkable work of Sir Kenneth Goadby (9, 10, 11), on latent infections in war wounds. Bacteria occur, isolated, in the solid matrix of sclerosed or repaired bone; and should not be disturbed, except when surrounded by, or enclosing, infected granulations, as in apical condensing osteitis or "loculi," constituting a real bone pathology.

Classes III and IV.—(III) Abraded or eroded teeth: occasional pulpal symptoms; (IV) average teeth: pulps diseased

It is in Classes III and IV (abraded, eroded and senile, coronally sound teeth: all conditions free from caries, infection, and gingival lesions, except clean gingival atrophy, which is not infective), that a most remarkable demonstration of the possibilities of the pulp as a reparative organ occurs. This is true up to a certain plane, after which, a gradual failure of function begins, the pulp displaying a singular inability to complete repair, so well begun, which results in complete degeneration.

This we recognize as inevitable, to be expected of this type of tissue, because of its peculiar vascularity, and its confined position and location as an end organ; but we must remember, according to Hopewell-Smith (21), "that nearly every degree of dentinal change is attended with hyperaemia and cell proliferation in the pulp tissue, and, generally speaking, formation of adventitious dentin." By tubular calcification and granular hyalin formation, the normal pulp responds successfully to erosive and abrasive dentinal invasion, supplementing this resistance against chemical and mechanical irritants by deposits of adventitious dentin on the approximating pulpal walls, or by pulp-nodule formation interstitially. This is typical normal defence, similar to that elicited in dental caries, but free from infection.

Such irritation in "one pulp may produce similar reactions in others of the same denture; pulp nodules are found at all ages and stages of all pulp disease" (26). Hence, such evidences of irritation are not definitely symptomatic. Degeneration may result at any time; and, except by referred pain or the radiograph, which are not definite, or by exploration and pulp removal, both destructive, we have no sure diagnostic methods, for thermal and faradic electric tests are misleading. Eventually, a series of symptoms of subapical disease arise in the periodontium, but these are periapical not pulpal.

With the advent of the gold inlay, applied to replace gold shell-crowns covering sound teeth ground down years ago, we have been given proof of Broomell's contention (5) that tubular calcification and much adventitious dentin result, possibly from dentinal irritation from

grinding, exposure to thermal change, or cements. In recent cavity preparations, areas of sensitive dentin are found in such teeth; the pulps are frequently degenerate, but little pulpitis or apical disease is present, except in those previously infected through deep carious cavities. Many such have been restored to full function by M. O. D. inlays. The shell-crown should, therefore, be condemned; if need be, not for the pulpitis it induces but for the gingival disease resulting from its careless use.

Arteriosclerosis is associated with pulp degeneration; but diseased pulp vessels are commonly sclerosed, without systemic hypertension symptoms. Advancing age is a real factor, these conditions being rare in the young. We know that senility induces, in sound teeth, fatty or mucoid degeneration of odontoblasts, the entire pulp becoming fibrotic, sclerosed, or partially or completely calcified. Any of these conditions may end in gangrene, infectious or otherwise. But we know no better way of determining the progress of this course of events, inside the tooth, save by appearance, than we have in the patient from his history and facies.

It has been the practice not only to blame pulp degeneration for causing periapical disease, but also to remove such pulps in the hope of preventing the putrefactive (not infective) products of degeneration from causing such disease. It has also been the fashion to speak of hematogenous pulp infection in these groups, and also in coronally sound teeth with the pulps non-vital from trauma. In the light of quoted research, neither practice nor theory in these respects is tenable. It is more than likely that nearly all periapical disease, except that produced by the dentist chemically—by radical canal medication—results from the arrival in the pulp of a real infection via the dentinal tubuli or by pulp exposure, or via the gingival lymph vessels, producing chronic pulpitis, the toxic products of which in turn produce periapical infection. And it seems that apical disease is just as rarely produced by chemical products of pulp degeneration, minus infection, as is pulpal infection, in sound teeth, the result of hematogenous convey.

The pulps of coronally sound teeth, which are necrosed or gangrenous, present necrotic masses entirely devoid of normal tissue-elements, and may become infected, involving the apex at any time,

with no pulpal but many peridontal symptoms. This process is slow, however, and such gangrene is often walled off, even in the dental pulp, according to Witzel and Arkövy, by a "line of demarcation" of granulation tissue, the subgingival remainder being in a passive (venous) hyperemic state, when vascular stasis usually results, with less liability of toxicity appearing beyond the foramina before the pulp can be removed.

Until better diagnostic methods are devised, we deduce the following:

Conclusion 4. Unless pulp symptoms or exposure arise in eroded, abraded or senile teeth, it is meddlesome practice to risk pulp devitalization and defective canal filling, just because pulps *might* degenerate, the exception to this rule being pyorrheal teeth, which might be infected by the gingival pathway.

In all stages of degeneration, except the calcific, when the pulp is finally infected by opening the pulp cavity or otherwise, periapical disease is both rapid and virulent. This is to be expected and illustrates a lack of the usual resistance, resident in healthy dentin and pulp; for pulp cells are fibrotic, fatty or gangrenous, as are the fibrillae. The entire tooth is thus a perfect culture tube.

Conclusion 5. Cases in Class IV (fresh surgical exposure of symptomless pulps, made through hard dentin, even at the risk of its being infected) are hopeful. Capping should be attempted, with frequent clinical and radiographic survey; but the slightest periodontal symptoms demand immediate pulp removal. This is especially true of young unfinished apices, which should be considered with a full understanding of the results of the recent research on apical cementum by Box (3).

No pulp exposure with the least symptom of any disease, except calcific degeneration, should be capped, for periapical infection is inevitable, insidious, and symptomless, hence dangerous.

Class V.—Carious teeth; pulps infected

Class V (carious teeth, infected pulps, but subgingival dentin not completely infected, with the apices free from disease and vital) is most interesting, because it is in this group that the profession, in the past, has done its best work.

In spite of infected and defective canal work and so-called "useless medication" (before the advent of the radiograph), it can be shown that a large percentage of such teeth were preserved, and are harmless and functional today, notwithstanding the fact that the pulpitides of this group are the principal portals for the production of periapical disease, and that pulp diagnosis was crude and inaccurate. The errors of the past lay not alone in septic canal work, nor in empirical treatment, but in the empirical dictum that every tooth must be saved at all hazards, and also in failures of both treatment and judgment in the selection of teeth for treatment, no distinction having been made between those of Class V, with vital apices, and of Class VI, which were hopelessly abscessed.

It is always instructive, even if humiliating, to review past failures. In the following statistics, instances of periapical disease, diagnosed clinically and by radiograph, are rated as failures, a percentage of which no doubt occurred prior to treatment and should appear under apically diseased teeth in Class VI. The number of rarefactions, mistaken for real bone lesions, would likewise greatly lower these ratios in favor of the apically well-filled pulpless tooth.

In all data submitted, the radiograph and its interpretation is a determining factor. While open to criticism, which will be discussed later, the radiograph, within clinical limitations, is the means by which the extensive occurrence of such lesions was first definitely discovered, not only about teeth, but in many other organs. It is still accepted as a most valuable diagnostic adjunct by the surgeon, so this is hardly the time for dentists to quibble over it.

Quoting from a previous compilation (16) of the results of the work of Callahan, Grove, Hyatt, and the writer, we find that only 7 per cent of root apices were ever perfectly filled and 53 per cent of these were periapically diseased. But of this 7 per cent, only 2 per cent showed filling material in the accessory foramina. Yet we know that these, as well as apical deflections, occur in at least 40 per cent of all teeth. Hence, in 38 per cent of "perfectly" (?) filled apices, the accessory foramina were not filled by filling material, but by pulp-shreds and vessels, vital or non-vital. Further, available filling materials (gutta percha usually) contract at body-temperature at least one-fourth in bulk (28), allowing seepage of exudates and bacteria. So, very few apices, in all their ramifications, have ever been filled.

In the same report, consider the data for poorly filled roots (fillings ending short of the apical third): 43 per cent of these occurred, but only 57 per cent were periapically diseased; and these, like the perfectly filled, may have been diseased before filling. Thus, we find approximately one half of both perfectly and imperfectly filled roots periapically diseased.

The second compilation is from Arthur Black's (2) "summary of abscesses related to root fillings," a report on six thousand dental radiographs of six hundred unselected adults. It is more fairly divided into percentages of small and large roots. It defines a good root-filling as one appearing well into the apex, with no open canal beyond it; a bad root-filling as not reaching the apex, projecting beyond it, or permitting the existence of a definitely open canal beyond the filling. In this report, 45 per cent of all filled roots were periapically diseased but only 9 per cent of these occurred about good root-fillings, while 63 per cent involved poorly filled apices.

All of this is most encouraging to those who would save and not extract the teeth of Class V. The 50 per cent of the first report and the 45 per cent of the second report, average 47.5 per cent of all filled roots as apically diseased, leaving a balance of 52.5 per cent not visibly diseased.

What of this 52.5 per cent? If it does not represent a composite of the patient's general resistance, and a local resistance existing in and about the tooth, plus average dental skill and effort in the past, what does it indicate? Who, and what, retained these teeth in function to the patient's great comfort, if the conservative dentist did not? Surely not the "100 per cent vitality men," both doctors and dentists, who extract all filled pulpless teeth. We are not disputing the fact that the remaining 47.5 per cent, apically diseased, were a menace to the patient's maxillae and adjacent teeth, if not to his general health, for undoubtedly they were and should have been removed immediately. But, in taking thought, it is good to realize that, by modern diagnostic, clinical, and surgical methods, even a much larger percentage of the teeth in Class V may be prevented from drifting into Class VI.

Any attempt to explain this 52.5 per cent of pulpless teeth, that apparently were not periapically diseased, leads immediately into

intricate questions of pulp pathology and symptomatology that can be discussed only clinically at this time.

It is recognized that various stages of pulpitis merge into each other more or less gradually and that these pathological phases may be fairly accurately defined in the laboratory, with a pulp slide in hand; but no associated definite set of symptoms differentiate such conditions in the pulp in situ. "Relative response to thermal change, dentin appearance and sensation, indefinite thermal and electric tests, or pain principally, which is rarely localized, appear to be our all, symptomatically, as indicated by the pulp. . . . . When sepsis passes out the apex, the damage we would avert has been done, and an outspoken group of symptoms present, but these are periapical, not pulpal. . . . . This is well illustrated by the treatment for pulpitis, which, notwithstanding a voluminous pathology, with few exceptions, is still pulp destruction" (16). If pulp diagnosis were accurate, periapical disease could be prevented, but with our present knowledge, "the treatment, pulp removal, unfortunately anticipates and is the pulp diagnosis" (16).

With this understanding, the prevention of periapical infection, by assisting normal tissue resistance and causing delay in extension of infection to the apex, by prompt pulp surgery and proper treatment, becomes a matter of the greatest importance. The first line of dentinal defence has been explained; it is frequently adequate, otherwise all carious and filled teeth would contain septic pulps, since caries is partially a septic process. The second, occurs interstitially after actual pulp invasion. This resistance is best described by Hopewell-Smith (21), who says: "It is not at all surprising that this is a structure, which possesses great recuperative power, and is constantly exercising its functions in this respect, and undergoing repair; hence, any invasion by disease or the occurrence of an accident is succeeded by a resistance, which, in many cases, is highly satisfactory."

The chances for apical infection can best be grasped, by a comprehension of Prinz's (29) classification of pulpitides into (a) those which are *rapid*—the acute, destructive types, i.e., acute, simple, and purulent pulpitis; and (b) those which are *slow*—the chronic, productive types, i.e., chronic ulcerative and hypertrophic pulpitis. In any clinical discussion of pulpitides, Noyes's remark in this

relation must always be kept in mind. He says (26): "There is the greatest difference in the rapidity with which these stages follow each other and the extent to which the inflammation spreads through the tissue before the breaking down begins. This is probably due to the character of the invading organism and the resistance of the individual."

In acute destructive types, the entire pulp may rapidly become purulent or liquefied, odontoblasts and fibrils may become necrotic, and complete dentinal and apico-cemental infection occur, when acute or chronic apical diseases quickly follow. Or, thrombosis and infarct may induce gangrene, which has been described as slowly involving the apex; or both phases may be subacute, and result in chronic ulcerative or hyperplastic stages. But, as a rule, acute pulpitis is dangerous, producing apical disease before the pulp can be removed. In chronic productive types, the situation is much more hopeful, and dentinal and pulpal resistance more active; for the odontoblasts and fibrils are not quickly liquefied and protect the subgingival dentin for some time. Coronally, infection may progress between the odontoblasts and pulpal walls, but subgingivally and apically this layer remains well attached and more or less vital.

Below the gingivae, infection primarily follows a central course along the great vessels, attacking the pulpal-wall tissue later. Occasionally, the perivascular lymphatics convey bacteria and toxins through the foramina, producing periapical disease, while the pulp is still sensitive and vascular, and the apex vital—a very dangerous condition. Usually, however, there is much delay, and time enough to prevent apical disease by pulp removal. Particularly is this true of hyperplastic types, where a productive cell reaction, with much granulation tissue often covered by epithelium (as in pyorrhea), is associated with pulpal tumor. Resistance to infection is high in, and infection is slow in passing through, foramina; in these, prompt clean canal surgery should leave an uninfected vital apex and functional tooth.

The following deductions are, therefore, justified, from the standpoint of histopathology, as relating to conditions that guard the apex and prevent or delay its infection.

(A) In chronic pulpitis, and certain stages of pulp degeneration and gangrene, complete destruction of the subgingival odontoblasts and fibrillae is usually a slow process. Persistence of this layer of cells, vitally attached to pulpal walls, has been noted by many histologists, for all pulp diseases. It guards the apical third of the dentin, possibly transmitting sensation, which often persists, indicating a vital apex. Such would not be the case, if this layer was necrotic or liquefied.

(B) Pulpal infection is generally localized centrally. Excepting the initial lesion, the carious exposure, it follows the large vessels and perivascular lymphatics centrally through the pulp, leaving tissues adjacent to dentin and cementum intact for some time. Indeed, this pathway is more accessible for the production of periapical disease than of general infection of the apex.

(C) The presence of nerves, under these pulp conditions, sufficiently nourished and functional to record pain, denotes a persistent vascularity. The common resistance to arsenic, cocain, and other medicaments, offered by many stages of chronic ulcerating and granulating pulpitides, is hopeful, and indicates activity of nature's usual granulation methods, protecting the apex and contained vessels against infection.

(D) The results of recent research, by Box (3), conclusively demonstrate a complete circulation of tissue fluids, through the cemental apex from the periodontium via the lacunae and canaliculi, the granular layer of Tomes and dentinal fibrillae, to the apical third of the dental pulp. This condition emphasizes the source from which the cemental matrix of a vital apex, and possibly apical pulp-shreds and vessels, are nourished; for they are thereby in touch with serologic factors, which not only fight infection, but prevent it. It is a most hopeful feature; but, when once this labyrinth is infected, these lines of communication become pathways by which sepsis passes into the apical periodontium, destroying and detaching it, and creating a thoroughly septic, necrotic, apex and periapical disease.

(E) There is the further fact that this means of communication cannot be demonstrated in middle-third cementum (3), the first circumferential lamella (lying next to the granular layer of dentin) of which is unorganized, with no connecting canaliculi crossing it

from pulp to periodontium. This is, indeed, a protective feature, for, if infection can be prevented from reaching the apical dento-cemental communication, a definite barrier exists against it in the middle and gingival thirds. If this were not true, alveolar abscesses would commonly occur beside the root on the middle third, as para-radicular, instead of periapically on the apical third.

What part had infected-canal methods of the past in preventing periapical disease in the 52.5 per cent of teeth that were free from it? And what may we expect of future canal surgery? It has been demonstrated by Brooks and Price (4) that dentin and cementum of experimental "dead" teeth in the laboratory, once thoroughly saturated with a known and virulent infection, cannot be sterilized, except by agents destructive of normal tissues. This is even more true of a tooth in situ, many statements to the contrary notwithstanding. If dentinal and apical cementum cannot be sterilized, and canal medication was worse than useless; if, as shown, very few apices have ever been filled, except by leaking fillings; then, all the canal work of this period was useless and all teeth so treated should be periapically diseased—yet this, happily, was not true in 52.5 per cent of the cases.

There is no doubt that the remaining 47.5 per cent were as thoroughly saturated with infection as the "dead" test-teeth of Brooks and Price, and that all treatments in these denuded and infected apices, surrounded by infected granulations, were as useless then, as we believe them to be now. There is much reason for believing that, in the other 52.5 per cent, a resistance inherent in the tissues, previously described, was so supported by canal medication and pulp-removal methods of the period, that (a) infection and putrefaction were delayed in subgingival dentin and apical-third pulp-shreds, and that (b) complete apical infection was prevented by germicides placed directly into the pulp tissue, which is quite impossible in deeply infected dentin. Dentin was not sterilized, but apical dentin and cementum were not permitted to become deeply infected by the removal of these much medicated pulps. Infection was thus confined to the middle root-third, with its impermeable cemental laminæ, until the apex and vulnerable apical dento-cemental communication could be filled.

We believe that, by modern modifications of this method, in which greater speed and less radical medication are necessary, many more Class-V cases may be saved from ultimate apical disease; hence:

> *Conclusion 6*—for the teeth of Classes I, II, III, IV and V, diagnosed carefully and promptly: "If pulps can be removed under as nearly aseptic conditions as possible, by careful chemico-mechanical instrumentation, leaving only inaccessible vessels and vital pulp-shreds in the immediate foraminal openings of a vital apex, which has not been cauterized nor over sterilized; or, if these strong agents can be used without their passing out into the membrane (though conservative operators prefer milder medicaments)—if all this can be accomplished without perforation and encapsulation, avoiding infection from debris, then, any canal-filling method, as nearly aseptic as can be, that will close these openings well into, but not through the apex, will insure apices infected as little, and filled as much, as possible and functional teeth" (16).

Any discussion of pulp removal raises the question of its apical boundaries. These are defined according to age. The young apex (papilla stage) presents one large trumpet-shaped foramen, with few cemental laminæ to or through which the pulp extends. It is next to impossible to remove such pulps or fill the foramen without producing periapical disease. In the adult apex (pulpal stage), which is rounded or pointed, and built entirely of cementum about the main and many accessory vessels, forming multiple foramina (40 per cent occur in all teeth), the pulp proper is only that tissue bounded by odontoblasts, as explained by Grove (17, 18), and the apex contains only vessels.

In all poorly filled, and 38 per cent of well filled apices, these foramina still remain unfilled, because no method, in or out of the mouth, is known for detecting their number or deflected course; and, even if detected, no efficient technic is known which will either safely remove the vessels or fill the foramina. So, the tissue generally removed, except in intentional perforation, which finds or makes, and fills, one opening, neglecting all others, is that bounded by odontoblasts.

"There is some evidence for believing that remaining vessels and apical pulp-shreds, lying in touch with surrounding vascularity, either become organized into fibrous tissue or foramina are closed by deposits of cementum or osteo-dentin" (16). This can occur only in a vital apex, not infected, nor saturated with chemicals, nor perforated and over filled; and only in one to which the periodontium is physiologically attached (27).

The vital apex (13, 14) is, thus, the crux of all canal operations. Its maintenance is worth any amount of time and effort. It cannot be encapsulated because periodontal fibers are everywhere attached to it. There is, therefore, no denudation or hypoplasia in which encapsulations may lie, unless they traumatically protrude into the membrane, granulomata, or cysts. Quite the reverse: the denuded apex, necrotic by whatever means, is not worth a moment's effort, no matter how medicated nor how well filled. One of the gravest mistakes of dentistry is the stubborn belief that correct root-canal filling will cure apical disease. The most perfect canal operation is never a curative, but only a preventive procedure. It will never cure, and often does not prevent periapical disease. The warning against over-medication too prolonged or too high in oxidizing power, should again be emphasized. "There is more occasion to fear chemical tissue-invasion, than infection, because bodily resistance is better prepared against infection than against chemical necrosis; particularly is this true of connective tissues" (13, 14, 15).

"If none of the known methods really fills apices, in all their ramifications, what has protected the subapical tissues and patient in the defective work of the past and present against infection, or reinfection? . . . . It is daily becoming more evident, to many investigators, that some process other than the dentist's processes, is the final equation in root canal treatment and filling. This factor which fills, and in the past always has filled, or repaired, foramina unfilled, in so-called perfectly filled roots (preventing infection in 52.5 per cent of imperfectly filled roots), is the complicated reparative process common to all body connective tissue, namely, leucocytic function (endothelial leucocytes, etc.) plus good fibrous tissue organized by fibroblasts, cementoblasts, etc. These investigators approve any method that improves apical technic, provided it does not interfere with this reparative function" (16).

All of the foregoing cannot be used by the operator as an excuse for negligent pulp removal, or in expectation that strong medication may be substituted for the finest canal technic—it should stimulate him to even greater effort, that repair may be induced free from infection and without radical medication.

There is an increasing group of radical operators who extract all pulpless teeth because they are infected. They should proceed to the ultimate, and remove all carious and pyorrheal teeth because, as we have shown, they also are infected, if not diseased. The mere presence of bacteria in a fibrous tissue is not disease, to which dignity it does not rise until certain well known cell-reactions occur. How are we to distinguish as between the periapically diseased tooth, where this has resulted in the subapical tissues, in touch with the blood stream (Class VI), and the apically well filled pulpless tooth, where it has not occurred (Class V)? The answer lies in correct diagnosis of apical disease, defining the normality of the attaching tissues, namely, cementum, periodontal membrane and alveoli; all equally as important as, if not more than, the pulp.

The laminated cementum is built by and in the fibrous periodontium. Both are developed according to age and use. The laminae and fibers of both respond to every stress throughout life by repair, which is not uncommon even in controlled infection. These are no more important, attaching the tooth, than the dense lining of the alveoli known as the lamina dura, which receives the insertion of the osteal ends of these fibers. If the cells of these tissues lie in physiologic apical contact, attaching a well filled tooth from which the pulp has been removed, that tooth is healthy, no matter whether it be called a "non-vital," "pulpless," or "dead," tooth (26). When this contact is broken, periapical infection exists beyond the tooth and can best be diagnosed by radiographic study, modified by clinical symptoms. "Principal among the clinical bony land-marks stands the character and stability of that hard lining of each alveolar socket known as the lamina dura. . . . . Showing as a dense line surrounding each tooth, uniting with its neighbor to form the interseptal crests, supported by trabeculae, which are important, it defines circumferentially the normal width of the periodontium. Its disappearance in areas, particularly in middle-

and apical-thirds, with thickening of the dark line representing the periodontium, indicates undue stress or proliferating periodontitis. It may be completely fused in this region by normal cemental repair, or the hyperplasia may progress to a hypercementosis. It is missing in cemental hypoplasia and denudation, or when the periodontium is destroyed in the midst of suppuration, granulomata, unorganized granulations or cysts. . . . . Lack of continuity in the lamina is undoubtedly a most valuable symptom of infection advancing beyond the tooth; and, clinically, if it be intact, the cementum should be considered healthy" (16).

"The radiograph taken and developed according to the latest technic by those familiar with facial and dental anatomy, in form generally of intra-oral films, sometimes extra-oral plates, is simply indispensable. . . . . Radiographs do not show infection, nor disease, of facial or mandibular bones and soft parts, any more than in other body tissues. They do show, by contrasting radiopacity and radiolucence in two dimensions, in all hard and many soft tissues (as no other method will), destructive, constructive and developmental variations, when compared with normal surrounding tissue" (16). Serious pararadicular disease is occasionally masked by roots paralleled or superimposed on the film; but periapical rarefaction, occurring around the apex, is always visible and diagnostic of a dead apex; about which the subapical bone can never form, for it is negatively chemotactile. Bone repair and sclerosis may occasionally present as dense as cortical bone, but the trabeculae and partitions of the adjacent normal bone, to which these should be compared, are missing.

"Sequestra occasionally seem more radiopaque than normal bone and rarefactions may result systemically from osteoporosis, osteomalacia, pregnancy, or calcic waste; in fact, many unusual factors may, but rarely do, appear. . . . . Exceptional cases cannot be compared with the usual, and should not be quoted, disturbing confidence of honest operators seeking help, nor can they be used by dishonest operators seeking an excuse" (16).

There can be no better illustration of the diagnostic accuracy of the radiograph in the radiographic study of all kinds of bones, than the striking regularity with which radiographic rarefaction defines apical

disease as indeed a strictly periapical and not a pararadicular disease, involving middle-third periodontium. The radiograph demonstrates, in thousands of films, the usual infectious portal through the root canal, via the apical dento-cemental communication of Box (3), and confirms the importance of this discovery in histopathology as the cause which limits all surrounding granulations, cysts, etc., strictly to this area. The constancy with which this occurs demonstrates the efficiency of the barrier offered against this (the usual) pathway by the impermeable first cemental lamina of the middle-third, with no communication between pulp and periodontium.

"If radiographs can be trusted in abnormal tissue study, they should be trusted to show normal tissue detail and repair; and if there be a defect, it is in showing too little rather than too much tissue involved. No better diagnostic means is at hand. The whole matter hinges on the clinical features, plus the operator's attitude in interpretation, which must never be attempted unless the patient's history and tissues are under direct survey" (16).

With the foregoing diagnostic, operative, and reparative possibilities in mind, we deduce the following:

> *Conclusion 7.* If pulp removal and canal filling can be accomplished according to the conditions specified in *Conclusion 6;* if the lamina dura is definite, and surrounding bone cancellations appear to be normal, as in adjacent vital teeth; if there are no clinical symptoms and the apex is not hypoplastic, but contains a filling well into, but not through it, with no visible open canal beyond; then, a pulpless tooth so situated should be retained, as it is functional and harmless.

Because of inoperable canals and multiple foramina, the superior molars and canines, and the inferior molars, are occasional exceptions to this rule, which should be kept under survey. If periodontal symptoms arise, the affected teeth should be promptly extracted.

It is true that a percentage of past canal-work, still remaining, is doubtful in quality and should be kept under observation; but there is no justification for suspecting the vital apex, filled by modern methods, except as noted.

It is distinctly not true that apically well-filled teeth of the past, showing no apical disease, are a menace, and should be extracted in

the ill patient, or as a diagnostic experiment in one who is under observation, just because periapical disease, even if not demonstrable, *might* sometime result.

Occasional cases are reported of the occurrence of apical disease on well-filled teeth, by hematogenous convey from other focal areas or systemic disease. Similarly, it is said to arise from chronicity or lowered resistance. The writer has observed this frequently in poorly filled, previously diseased, apices, the chronic condition becoming acute; but never except as noted in a well treated apex. Granted such a possibility: if well filled apices have not as yet been involved in a patient suffering from metastases, toxemia, or the reduced local and general resistance of chronic disease, the chances are that periapical disease will never so result; and such teeth should be retained, for no better test could be had of the apical repair and the success of the operation.

In the writer's experience and from a complete survey of the literature, very few well filled apices, free from apical disease, have ever been cultured with positive results. Quite the reverse: thousands of laboratory studies prove that all poorly filled and apically diseased teeth are infected or surrounded by infection. Hence:

> *Conclusion 8.* General nutrition may be seriously disturbed, and great distress caused the patient, by indiscriminate removal of Class-V (apically well-filled) teeth, for diagnostic purposes or because infection is suspected. Such operations are frequently peremptorily ordered by medical men in their anxiety to cure disease and in ignorance of oral pathology. Obviously, such teeth are extracted by a dentist, who, if he does not protest or refuse, but operates following a fad or through fear of, or to please, his medical confrère, undoubtedly damages the patient's health and lowers the dignity of his profession.

Necessarily there must be a real local pathology in the attaching tissues, a definite disease focus in touch with vascularity, before a general or systemic pathology can result from it. The term "focus" is most happily defined as a "fire-place," and such must really exist before its bacterial content may be raised in virulence, or its cell reactions permit of toxicity and general focal infection. This can and does occur in the improperly treated open canal, where the

entire tooth, as well as the surrounding tissue, may well be the "fireplace;" but not in a well-filled apex, protected by normal periodontium.

Class VI.—Infected teeth; septic pulps

There is little use in consuming time for a consideration of the teeth of Class VI (infected teeth, septic pulp, involved by periapical disease). They have been much discussed in former papers (12, 13, 14, 15, 16) and are really not worth while, for the reason that no adequate treatment is known which will certainly cure the suppurative, granulating, or cystic, phases of apical disease, except surgery. This statement implies recourse to (*a*) *apiectomy*, which is rarely successful, because the apical section of dentin and cementum, left by the operation, is not sterile, and generally, if such tissues are sterile, apical disease does not exist, hence there is no need for apiectomy; or (*b*) *extraction*, which obviously destroys the tooth, but does not cure the bone lesions, except when followed by (*c*) *curettement*, the third and most important surgical expedient.

A cure of periapical disease may be defined as regeneration of the lamina dura and associated trabeculae, comparable to adjacent areas on vital teeth or other operated areas, following extraction and curettement. It should result promptly (in four months) and the new bone should be deposited to a line the width of other periodontal membranes, in the same mouth, and the tooth must be clinically correct. Such repair is indeed rare. It has been noted occasionally in acute cases, where subapical tissues were sharply infected by acute purulent pulpitis, and were as quickly relieved of exudates by prompt drainage and canal treatment. These cases reverse the rule, which is that of a primary chronic infection, usually followed by an acute secondary parulis or fistulous abscess, associated with a necrotic apex. Any treatment of this latter type seldom succeeds; subapical bone may appear, as if repaired, but soon breaks down, invading a larger area. Hence:

Conclusion 9. With the exceptions mentioned, all periapically diseased teeth, when clinically and radiographically definite (Class VI), should be promptly extracted and sockets curetted. If there be the slightest question, it is best to err radically, and

remove a doubtful tooth, than to risk inevitable damage to adjacent teeth, attaching tissues, or the maxillae and possibly the patient's health.

Many medical men, principally pathologists, believe the oral focal-infection idea to be much overdone. Others do not subscribe to it at all. Whatever the outcome of this disagreement, we conclude as follows:

Conclusion 10. Dentists generally must surgically remove all such lesions, regardless of whether they be infected or not, because a real chronic bone pathology exists about the tooth and its cell reactions often simulate malignancy—it eventually proliferates, undermining the denture. So, obviously, the dentist, quite as urgently as the physician, and certainly the patient, desire prompt action to prevent further bodily invasion.

IV. CONCLUDING REMARKS

The writer is just as confirmed a believer in the doctrine that apically diseased teeth (Class VI) are infective foci, which may cause systemic disease, as he is in the fact that pulpless, apically well-filled, apically healthy teeth (Class V) do not produce systemic disease and should be retained. The relation of these lesions, to the various systemic diseases with which they are associated, has been discussed in previous papers (12, 13, 15). Quite recently it was ably presented to this society by Barker (1), whose statements, the writer, from experience in private and hospital practice, heartily endorses. It may interest you to learn, in this connection, that the results reported by Barker, in clearing up oral focal infection, were obtained by radically removing and curetting all pulpless teeth periapically diseased, and by leaving in the mouth all apically well filled pulpless teeth, not so diseased. Such results have been so obtained in the last seven years, with as many recoveries to the credit of this method as, if not more than, to the "100 per cent vitality" procedure. But it must also be noted that all other focal areas, elsewhere in the body, were likewise radically blotted out. Microscopic oral infection was not removed, and macroscopic bodily foci ignored. Thus, the focal "hobby" was not overridden.

The closure of oral infective portals, if attempted at all, should be uncompromising. Only by promptly ruling out all systemic foci will "the fight long continued against a minute but daily toxic invasion be won; and these principles should be applied equally to all who consult us, either sick or well" (15).

The dentist who, without medical assistance of the highest order, attempts to distinguish between the sick and well patient, ignores recent experience of the Medical Advisory Draft Boards, where it required ten specialists, of whom one was a dentist, to diagnose an ambulant man, to say nothing of a sick one. The dentist presumes by a glance and a few routine questions to determine, instantly, matters which might keep hospital laboratories busy for days. We dabble superficially in internal medicine, when we do not know the actual etiology of or sure treatment for a single dental disease.

One says the complement-fixation test for a streptococcus is as definite as a Wassermann test, when we know it is not; and proceeds to treat apical disease, retaining teeth when it is negative, and extracting when it is positive. Others speak vaguely of dyscrasias, diatheses, and susceptibility to streptococcus. Again another assures us that, if the tonsils are normal, the kidneys or heart and all blood counts correct, we may, without looking further, experiment with septic apices. Many criticise the internist for ordering out abscessed teeth, yet few profess actually to cure them; and, withal, none admit that they are internists. The circle of modern group-medicine always centers in the judicial diagnostic-mind of the skilled internist, who surveys the reports of all associated specialists, writes the final diagnosis and dictates the treatment.

The position of modern dentistry in this combination is now assured and is most honorable. We, of all specialists, are deeply interested, and should be most active, in clearing up all the present oral sepsis; and we are doubly interested in its prevention for the future.

If an amount of energy similar to that which is now being expended by many dentists, in heralding recovery from systemic disease caused by oral sepsis, could be put into the oral hygienic effort or into further research into dental caries and pyorrhea, for instance, we might eventually prevent these diseases and then there would be little oral sepsis

to treat. This would be a much more becoming and modest pose; and, furthermore, it would be attending to our own business—in our own field of pathology, of which we know so little.

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201 W. Madison St.

THE ORIGIN AND EVOLUTION OF THE HUMAN DENTITION[1]

A Palaeontological Review

(*Continued*)

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PART III. NATURE'S EARLIER EXPERIMENTS IN EVOLVING LARGE-EYED AND SHORT-JAWED PRIMATES[1]

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I. THE LORISES (LORISIDÆ)

The existing lorises, of Asia and Africa, and the galagos, of Africa, represent some of nature's experiments in evolving large-brained and short-jawed primates from a primitive lemuroid stock. Although they closely parallel the tarsioids in these features, as well as in the enlargement of the eyes and internal ears, their status as true lemuroids is established by the following characters: (1) their lower front teeth have assumed the peculiar lemurine specializations described in Part II of this review; (2) they still retain the primitive pointed

[1] Part I of this review was published on pages 89–193 (March), Part II on pages 215–283 (June), of this volume of the Journal of Dental Research.—(Ed.)

and projecting rhinarium, or snout, of the lemurs, which is replaced by a true nose in *Tarsius* and the higher primates (Pocock, 1918); (3) their hands and feet and (4) their reproductive organs (Pocock,

FIG. 77. THE SLOW LORIS (*Nyclicebus coucang*). AFTER D. G. ELLIOT, FROM A SPECIMEN IN THE NEW YORK ZOÖLOGICAL PARK

Habitat: Bengal, Burma. Suborder Lemuroidea, family Lorisidæ. One of nature's experiments in evolving large-eyed, short-jawed primates.

op. cit.) are much like those of typical lemurs, only further evolved in the same direction.

The chief lines of specialization in the skull and dentition of the Lorisidæ (*figs. 78–93*) have apparently been as follows: in connection

with nocturnal habits the orbits in the more typical forms have become greatly enlarged and their outer rims have grown forward, so that the eyes look partly forward and protrude widely from their sockets. Thus the orbits closely approach each other in the mid line, finally producing an extreme constriction of the interorbital region as in *Loris* (*figs. 79–91*). In correlation with the bulging and forward shifting of the eyes and the restriction of the interorbital space, the lacrymal bones become greatly reduced in size and the lacrymal ducts are displaced forward in front of the orbits. In spite of these specializations the whole bony face instead of being greatly retracted and bent down beneath the braincase, as in some higher primates, retains much of its primitive lemuroid appearance in which the face is wholly anterior to the low braincase, and the pointed insectivore-like snout projects strongly in front of the orbits.

Perhaps in correlation with the enlargement of the eyes and of the auditory parts of the brain, the cranium has become expanded laterally, especially in the occipital region (*figs. 86–93*). The middle part of the cranium between the posterior molars and the auditory bullæ appears relatively long, possibly in part because the orbits, in shifting forward, have carried with them the alveolar processes of the maxillæ. As the muzzle is produced anteriorly there is little evidence of crowding in the front part of the dental arch except when the upper canines are considerably enlarged, as in *Nycticebus natunæ* (*fig. 86*). The molar series in *Nycticebus* is restricted posteriorly, the third molars being small, a tendency shown in other groups of primates. The loss of p^1 in the upper jaw and p_1 in the lower jaw, on the other hand, was probably coincident with the transformation in the lower front teeth by which the canine was taken over into the incisor series, and the second lower premolar became enlarged and subcaniniform. This, in turn, may have been conditioned by the lemurid habit of cleaning the fur with the thick tongue and the lower front teeth. The lorises are said to feed upon leaves and young shoots, fruits, insects, birds and bird eggs, and for this mixed diet their sharp piercing canines and low-cusped cheek teeth appear well adapted.

The genera *Nycticebus*, *Loris*, *Arctocebus* and *Perodicticus* appear to form a structural series (*figs. 86–89*), showing a gradual loss of more primitive characters in the cheek teeth.

FIG. 79. *Loris tardigradus*, × 3/2

FIG. 78. *Nycticebus natunæ*, × 3/2

FIGS. 78 TO 93, INCLUSIVE. COMPARATIVE SERIES: SKULLS OF RECENT LORISIDÆ

Photographs by A. E. Anderson, figures rearranged from D. G. Elliot's monograph on the Primates. Plates loaned by the American Museum of Natural History.

FIG. 81. *Perodicticus potto*, × 1

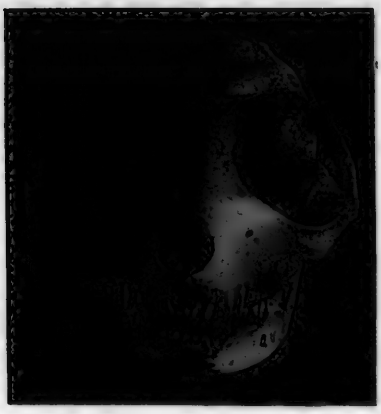

FIG. 80. *Arctocebus calabarensis*, × 3/2

FIG. 83. *Loris tardigradus*, × 3/2

FIG. 82. *Nycticebus natunæ*, × 3/2

FIG. 85. *Perodicticus potto*, × 1

FIG. 84. *Arctocebus calabarensis*, × 3/2

FIG. 87. *Loris tardigradus*, × 3/2

FIG. 86. *Nycticebus natuna*, × 3/2

FIG. 89. *Perodicticus potto*, × 1

FIG. 88. *Arctocebus calabarensis*, × 3 /2

FIG. 91. *Loris tardigradus*, × 3/2

FIG. 90. *Nycticebus natuna*, × 3/2

FIG. 93. *Perodicticus potto*, × 1

FIG. 92. *Arctocebus calabarensis*, × 3/2

Nycticebus. The dentition of *Nycticebus natunæ* is relatively primitive in the fact that p^3 has a well-developed internal cusp like that in the Eocene lemurs, while p^4 likewise retains a well developed postero-internal cingulum and sharp external cingulum, parastyle and metastyle. The molars also retain some of the crescentic or V-shaped form of the principal cusps usually found in Eocene primates. The proto- and metaconules are also distinct, and except on m^3 they have a well-developed cingulum-hypocone. The upper incisors, on the other hand, are often quite small. The canines are large and dagger-like with a pronounced worn surface on the postero-lingual face for articulation with the high, caniniform lower premolar (p_2).

Arctocebus. This genus is more or less intermediate in skull and dental characters between *Nycticebus* and *Loris*, on the one hand, and *Perodicticus* on the other.

Perodicticus. As compared with other lorises this retrogressively specialized genus has comparatively small orbits, a coarse, wider muzzle, stouter zygomatic arches and lower jaw, and a less voluminous brain case. The incisors and canines exhibit the typical lemuroid specializations, being procumbent and styloid. The lower anterior premolar (p_2) is enlarged and more or less caniniform. In the upper dentition there are three premolars, only the first of the primitive placental series being absent. The upper canine is considerably enlarged and dagger-like, directed vertically downward, and adapted perhaps to piercing fruit and to killing insects and small animals. The first functional upper premolar (homologous with p^2) is somewhat large and partly resembles the canine, since its border articulated with the subcaniniform p_2. P^3 is quite small, with very simple rounded crown, which may safely be regarded as degenerate in form. The rounded fourth upper premolar is bicuspid, so that p^4 and p^3 are very unlike. The first and second upper molars are tritubercular, with rounded cusps lacking external cingula and with very feeble hypocones.

The rounded form of all the cheek teeth is certainly a degenerate and secondary character. It has been paralleled in other phyla of mammals, as among the Viverridæ, Procyonidæ, Lemuridæ (*Chirogale, Attililemur*), Cebidæ, Hapalidæ. Similarly in the lower teeth

of *Perodicticus* the cusps are for the most part blunt and rounded. This retrogressive rounding of the teeth is also observed in modern man and to a certain extent in the chimpanzee, and is often connected with omnivorous habits. In *Perodicticus* the thickened form of the zygomatic arch, the widening of the muzzle, and the relatively small size of the orbits and braincase are probably all degenerative specializations, derived, through the conditions preserved in *Arctocebus*, from the more primitive conditions of *Nycticebus*.

Thus, careful and repeated studies of the skulls and dentitions of recent lorises and galagos lead to the provisional conclusion that on the whole the least specialized dentition is that of *Nycticebus*. Considering the dentition of the whole group, degeneration, or the loss of primitive characters, in this case, as in many others, has brought about an apparent simplicity or primitiveness in the end stages, which is at first sight very deceptive and likely to lead to erroneous phylogenetic conclusions.

Unfortunately but little or nothing is known of the palaeontological history of the Lorisidæ. *Pronycticebus*, of the Bartonian or Middle Eocene of France, represented by a skull (*fig. 133*) with well preserved cheek teeth, was regarded by its discoverer, Grandidier (1905), as at least structurally ancestral to the existing *Nycticebus*; in many respects it is certainly more primitive than that animal and stands on a lower stage of evolution. Stehlin, on the other hand, in his great work on the primates of the Swiss Eocene (1916, pp. 1421–1423), shows that *Pronycticebus* (*fig. 132*) resembles *Anchomomys* (*figs. 128, 129, 130*) in many characters of the dentition; and thus it may be connected with the stem of the tarsioid series (see below, page 393). *Pseudoloris* (*figs. 126, 127*), another primate of Bartonian age, also appears to be related to the Eocene tarsioids rather than to the lorises (Stehlin, *op. cit.*, pp. 1396–1400).

In conclusion, the Lorisidæ in their cranial and dental construction carry further the normal line of lemuroid evolution indicated in some of the smaller Lemuridæ, such as Microcebus and Chirogale, which they closely resemble in many characters. Their evolutionary trends contrast widely with those of many of the higher primates, in which the shortening of the face and its retraction beneath the braincase foreshadow the human conditions. So, too, the dentition of the Lorisidæ as a whole, although

sometimes retrogressive in the molar region, parallels that of the smaller lemurs and to a certain extent that of the tarsioids, but contains no promise of human possibilities.

II. THE GALAGOS (GALAGIDÆ)

The galagos were formerly united with the lorises in the family Lorisidæ, but they differ very widely from them in the elongation of

FIG. 94. HEMIGALAGO DEMIDOFFI. WEST AND CENTRAL AFRICA. AFTER ELLIOT
Suborder Lemuroidea, family Galagidæ

the tarsus and in the more *Tarsius*-like general appearance (*fig. 94*), so that they have lately been set apart as a separate family (Pocock, 1918). Their fossil history is entirely unknown.

FIG. 95. *Hemigalago demidoffi*, × 2

FIG. 96. *Galago alleni*, × 3/2

FIG. 97. *Galago crassicaudatus*, × 1

FIG. 98. *Galago elegantulus*, × 3/2

FIGS. 95 TO 110, INCLUSIVE. COMPARATIVE SERIES: SKULLS OF RECENT GALAGIDÆ

Photographs by A. E. Anderson; skulls after Elliot, rearranged. Plates loaned by the American Museum of Natural History.

FIG. 100. *Galago alleni*, × 3/2

FIG. 102. *Galago elegantulus*, × 3/2

FIG. 99. *Hemigalago demidoffi*, × 2

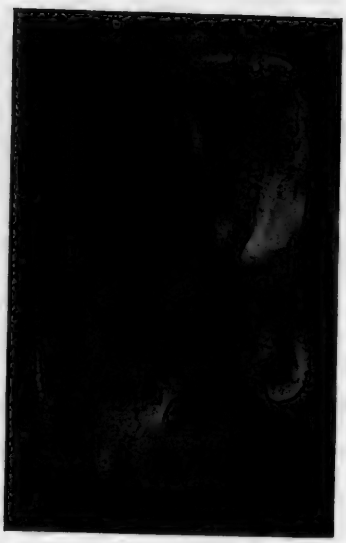

FIG. 101. *Galago crassicaudatus*, × 1

FIG. 103. *Hemigalago demidoffi*, × 2

FIG. 104. *Galago alleni*, × 3/2

FIG. 105. *Galago crassicaudatus*, × 1

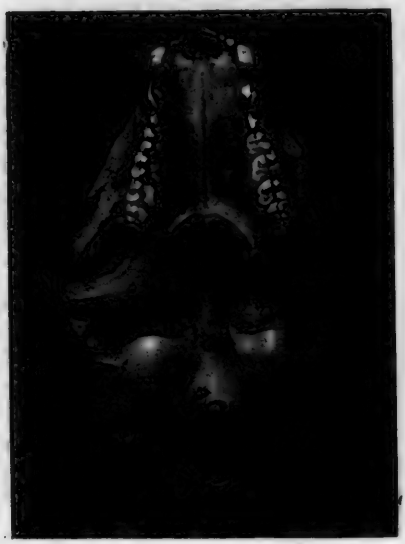

FIG. 106. *Galago elegantulus*, × 3/2

FIG. 107. *Hemigalago demidoffi*, × 2

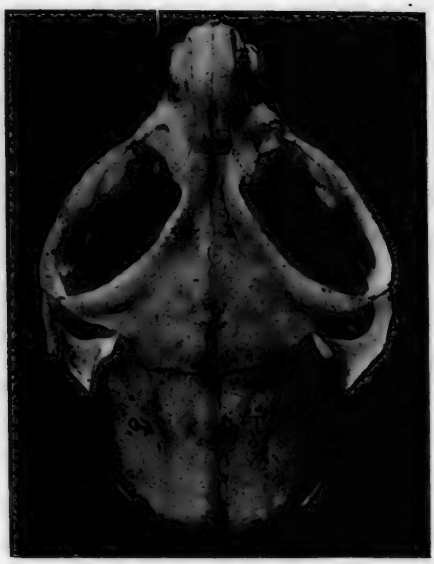

FIG. 108. *Galago alleni*, × 3/2

FIG. 109. *Galago crassicaudatus*, × 1

FIG. 110. *Galago elegantulus*, × 3/2

They are essentially nocturnal in habits and accordingly they parallel the Lorisidæ and Tarsiidæ in the enlargement of the orbits, in the restriction of the interorbital region, in the pointed elongate form of the muzzle, and in the transverse expansion of the braincase. But they differ from the typical Lorisidæ in the following skull characters: (1) the orbits (*figs. 107–110*) are directed more outward and are usually even larger than in the Lorisidæ; (2) the orbital rims and zygomatic arches are extremely delicate; (3) the cranium is not flattened posteriorly but is decidedly rounded across the occiput, and the mastoids, although inflated, do not project laterally; (4) the basicranial region (*figs. 102–105*) is often longer and the midcranial region relatively shorter; (5) the palate and dental arch are often narrower; (6) the mandible (*figs. 99–102*) is longer and much more slender and the ascending ramus is narrower. The skull as a whole is longer and narrower, with a more elongate muzzle and with the face even more produced in front of the cranium.

There are no very important differences between the galagos and the lorises in their lemur-like incisors and canines (*fig. 111*), but the fourth premolars of the galagos are submolariform, with a well developed cingulum-hypocone and two external cusps. Similarly, the fourth lower premolars are more advanced and submolariform, with well developed talonids, while those in the Lorisidæ are compressed anteroposteriorly, with reduced talonids. The upper molars are of the narrow tritubercular type, often with conspicuous posterior projections in the region of the metastyles and the upper molars with prominent cingulum-hypocones. They feed upon a mixed diet including fruits, insects, and small birds and their eggs (Lydekker).

Galago elegantulus (*figs. 98, 102, 106*) and *Hemigalago* (*figs. 95, 99, 103*) have extremely large eyes and well rounded crania, so that they especially suggest the tarsioids, but they retain the projecting nose and many other lemuroid characters (Pocock, 1918).

Thus the galagos are unquestionably true lemuroids, which parallel the *Tarsius* group, but are not ancestral to it. Nevertheless they illustrate, in certain characters, some of the structural stages by which the tarsioid grade of evolution was derived from that of the primitive lemuroids.

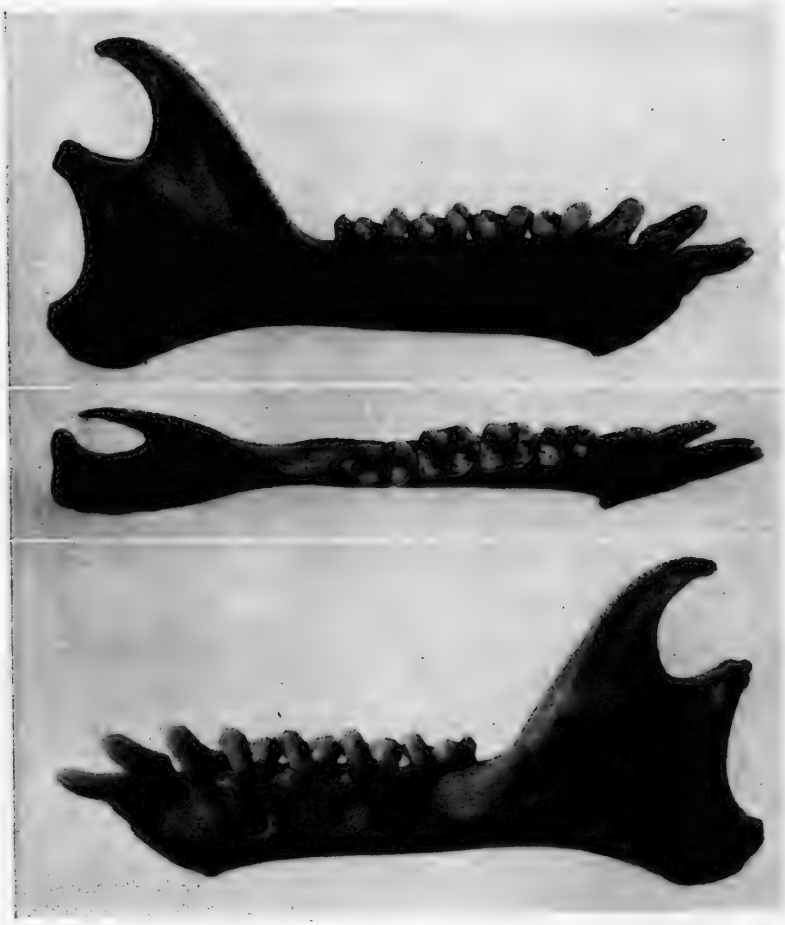

FIG. 111. GALAGO CRASSICAUDATUS. THREE VIEWS OF LEFT MANDIBLE. × 2. U. S. NAT. MUS., NO. 49,548

Suborder Lemuroidea, family Galagidæ. Shows the lemur-like specialization of the lower front teeth, the submolariform type of p_4 and the relatively advanced stage of the molars.

III. THE TARSIOIDS (TARSIOIDEA)

NORTH AMERICAN EOCENE TARSIOIDS

The Eocene tarsioids of North America have been described especially by Cope (1884), Osborn (1903), Wortman (1904), and Matthew (1915). They include a number of early specialized side lines of the Lower and Middle Eocene, such as *Tetonius*, *Absarokius*, *Anaptomorphus*, *Uintanius*, together with somewhat less specialized and more central but on the whole later genera of the Middle Eocene, especially *Omomys* and *Hemiacodon*. They are not known in later formations. On account of their various specializations none of them seems to be directly ancestral to the modern *Tarsius*, which is more probably derived from some Eurasiatic Eocene member of the group. They are all small and some are very minute in size, one of them (*Tetonius musculus* Matthew) having the three lower molars not more than 5 mm. in length. From the fairly well preserved skull of *Tetonius homunculus*, noted below (*fig. 118*), and from fragmentary remains of the skull in other genera, it appears that the American Eocene tarsioids, like their contemporary relatives in Europe, had very large orbits and were possibly nocturnal in habits, like the existing lorises, galagos and *Tarsius*; also that the braincase was widely expanded transversely, with slight or no sagittal crest. The jaws were slender (*fig. 112*) to deep (*fig. 119*) in front, the latter in those with enlarged front teeth; lower front teeth variously modified, but often with a pair of enlarged proclivous lower canines or incisors (*fig. 118*); premolar series reduced, the third and fourth upper premolars (*figs. 112, 116, 117, 119, 125*) tending to become bicuspid, the fourth lower premolar (*figs. 112, 118, 125*) often with high compressed crown; upper molars triangular (*figs. 112, 113, 116, 117, 119, 125*), wide transversely, in correlation with transverse widening of the talonids of the lower molars (*figs. 119, 124*); trigonids small, high; talonids low and large. Olfactory parts of skull much restricted; optic and auditory parts greatly enlarged (*fig. 123*). Limbs, so far as known, adapted for leaping in the trees, as in the modern *Tarsius*. This animal is said to feed upon insects and small reptiles, but apparently not upon fruits (Lydekker).

The known American Eocene tarsioids were rather widely separated from the contemporary lemuroids, both in dentition and in skull characters.

Omomys. Apparently the least specialized of the American forms is the genus *Omomys* Leidy (*fig. 112*), from the Lower and Middle Eocene of Wyoming. As described by Wortman (1904) and by Matthew (1915), the dental formula is $I\frac{?}{2}\,C\frac{?}{1}\,P\frac{3}{3}\,M\frac{3}{3}$ which, so far as known, is identical with that of the South American monkeys. The lower incisors are procumbent and the medial pair somewhat enlarged, the second small. The lower canine also is semi-procumbent, tending to become like the incisors. Thus even in this early genus there is already a tendency toward the enlargement and procumbency of the medial and lower front teeth, a feature which in some tarsioids finally results in an almost rodent-like condition.

The first lower premolar of the primitive primates has already been eliminated. The second has a pointed conical tip, the third (p_3) has a high, pointed protoconid, a triangular base, with an incipient metaconid; it also has a very small and crowded talonid overlapped by the front part of p_4. In p_4 the crown is submolariform, as there is a trigonid with an incipient paraconid and well developed metaconid in addition to the high protoconid; but the talonid is very small and crowded, and is widely overlapped by m_1, in correlation with the anteroposterior elongation of the lower molars. Thus there is a greater contrast between p_4 and m_1 than there is in the more primitive contemporary primates of the family Notharctidæ (described in Part II), and very possibly the abbreviation of the talonid of p_4 is partly secondary in this family.

The lower molars are fairly primitive, with a high, well defined trigonid and a comparatively low talonid. But the hypoconid is unusually large and projects buccally. This is correlated with the transverse widening of the upper molars and with the displacement of the para- and metacones toward the buccal margin of the crown. (Analogous instances have been cited in Part II of this work.) The third lower molar has a reduced trigonid and a fairly large median cusp, or hypoconulid. In general the lower dentition, even of this relatively primitive member of the tarsioid series, is probably more specialized than that of the oldest Notharctidæ described in Part II.

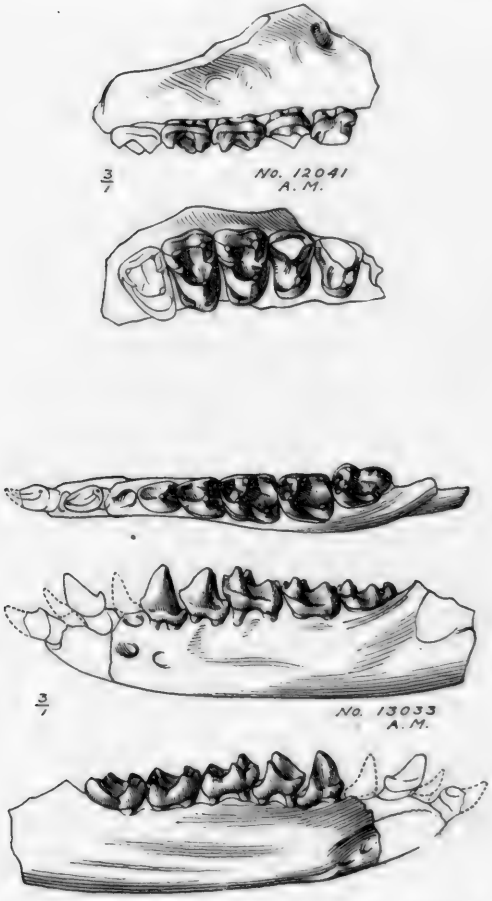

FIG. 112. *Omomys* sp. UPPER AND LOWER JAWS. × 3. AFTER W. D. MATTHEW

Upper jaw, outer and crown views. Upper Bridger beds (Middle Eocene), Bridger basin, Wyoming. Lower jaw, inner, outer and crown views. Lower Bridger beds (Middle Eocene), Bridger basin, Wyoming. Front teeth restored from Amer. Mus. no. 12,600. Suborder Tarsioidea, family Tarsiidæ.

The upper incisors and canines of *Omomys* are not known. The first premolar was probably wanting and p^2 was certainly small, as shown by its alveolus. P^3 is bicuspid and like p^4, but less advanced. It has a high, pointed apex on the outer side of the crown and a low lingual cusp continuous with the internal cingulum. P^4 is fully bicuspid, with a very prominent piercing external apex. Thus the tendency for p^3 and p^4 to become closely alike is evident even in the most primitive of this family.

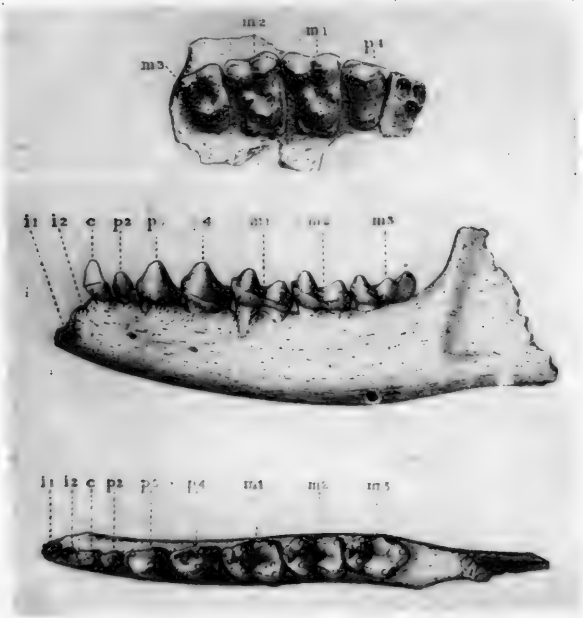

FIG. 113. *Hemiacodon gracilis*. LOWER JAW (LEFT RAMUS) AND RIGHT UPPER CHEEK TEETH. × 5/2. AFTER WORTMAN

Upper Bridger beds (Middle Eocene), Bridger basin, Wyoming. Suborder Tarsioidea, family Tarsiidæ.

The upper molars of *Omomys* are wide, in correlation with the width of the talonid. The external cingulum is reduced and the para- and metacones are more or less rounded, all relatively advanced characters. Well defined V-shaped proto- and metaconules are present on the sharp slopes of the protocones and have the usual relations with

the parts of the lower molars. The interdental spaces are well marked, in correlation with the comparatively primitive condition of the trigonids and the feeble development of the hypocones. They are retained throughout the family and even in the modern *Tarsius*.

Hemiacodon. This genus, from the Middle Eocene of Wyoming, is closely related to *Omomys* and may be regarded as a derivative of it

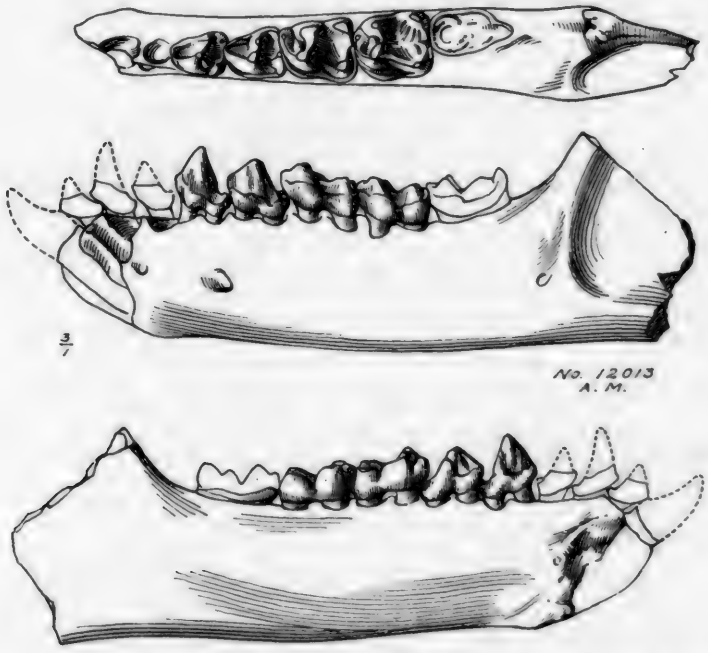

FIG. 114. *Hemiacodon gracilis*. LOWER JAW (LEFT RAMUS). × 3. AFTER MATTHEW

Upper Bridger beds (Middle Eocene), Bridger basin, Wyoming. Front teeth restored from Amer. Mus. no. 12,037. Suborder Tarsioidea, family Tarsiidæ.

(Matthew, 1915, p. 451). The wide upper molars (*fig. 113*) have large rounded proto- and metaconules and two small accessory cusps (proto- and hypostyles) on the internal cingulum of m^1, m^2. The third upper molar is much smaller than the others; the enamel surface of the crowns is wrinkled. The lower premolars, p_4, p_3, are somewhat but not greatly enlarged, and the lower front teeth are gently procumbent (*fig. 114*). The lower molars m_1, m_2 (*figs. 113, 114*) have

the wide talonids, characteristic of the group, and correlated with the great width of the upper molars. The lower front teeth, or central incisors, are semi-procumbent and considerably enlarged; the canines are small (*fig. 114*).

With regard to skull characters Wortman states (1904, p. 137) that in the fragment of a maxillary which he figures (*fig. 113*), "the anterior limits of the malar can be easily made out. It is thus shown that it does not reach forward to the lachrymal, but leaves the maxillary a considerable share in the anterior boundary of the orbit, as in the monkeys. The rather small, single, infraorbital foramen is situated above and opposite the posterior edge of the third premolar, about in the same relative position as that of the squirrel monkey. The maxillary gives further evidence of proportionately large orbits, and if the superior dental formula was the same as that for the lower jaw, the muzzle must have been considerably abbreviated. The whole aspect is, in fact, not only characteristically Primate, but one considerably advanced."

Washakius. As described by Leidy (1873), Wortman (1904), and Matthew (1915), *Washakius* (*figs. 115, 116*) is another very small tarsioid from the Middle Eocene of Wyoming, which has the dental formula $I\frac{2}{2}$ $C\frac{1}{1}$ $P\frac{3}{3}$ $M\frac{3}{3}$. *Washakius* is distinguished especially by the presence of metastylids (accessory cusps posterolingual to the metaconids of the lower molars), and by the coarse wrinkling of the enamel in the upper molars. The latter have the main cusps well rounded rather than angulate, and the first and second upper molars have small but rounded hypocones. The lower incisors were small, much less procumbent than in *Hemiacodon;* the lower canine was small, not procumbent. From the shape of the superior maxilla, figured by Wortman (1904, p. 210), it seems very likely that the orbit was large, as it was in *Hemiacodon* and in the older related genus *Shoshonius* (*fig. 117*), which is distinguished especially by its large mesostyles and by the feeble development of the internal cingulum cusps.

Tetonius. That the tarsioid group very early became separated from the lower lemuroid primates is indicated by the fact that even in the Lower Eocene it is already represented by a relatively specialized member, *Tetonius homunculus* (*figs. 118, 119, 121, 123*), the *"Anaptomorphus" homunculus* of Cope. In this animal, as lately

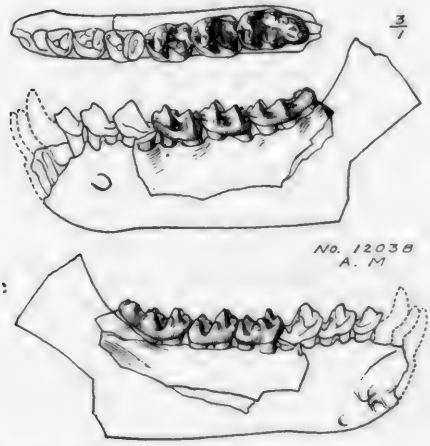

FIG. 115. *Washakius insignis*. LOWER JAW (LEFT RAMUS). × 3. AFTER MATTHEW

Upper Bridger beds (Middle Eocene), Bridger basin, Wyoming. Suborder Tarsioidea, family Tarsiidæ.

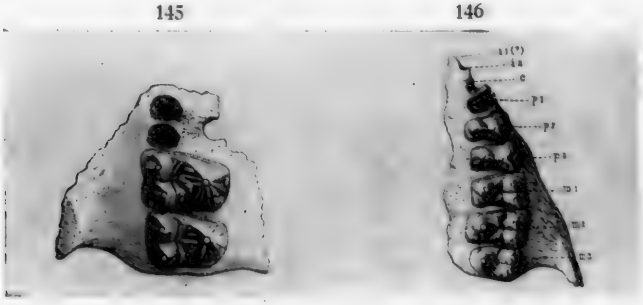

FIG. 116. *Washakius insignis*. UPPER PREMOLARS AND MOLARS. AFTER WORTMAN

145.—Second and third right upper molars and fragment of maxilla. × 4.

146.—Left upper jaw with p^3–m^3. × 3/2. Upper Bridger beds (Middle Eocene), Bridger basin, Wyoming. Suborder Tarsioidea, family Tarsiidæ.

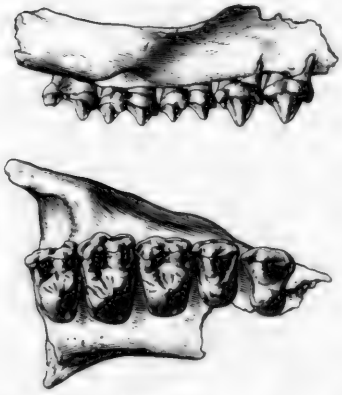

FIG. 117. *Shoshonius cooperi*. RIGHT MAXILLA. × 4. AFTER GRANGER

Wind River beds (Lower Eocene), near Lost Cabin, Wyoming. Suborder Tarsioidea, family Tarsiidæ.

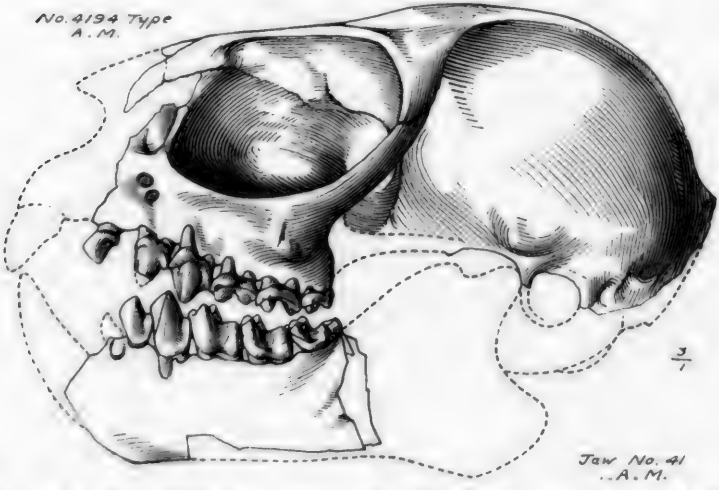

FIG. 118. *Tetonius homunculus*. SKULL AND LOWER JAW. × 3. AFTER MATTHEW

Distortion of skull corrected. Gray Bull beds (Lower Eocene), Bighorn basin, Wyoming. Lower jaw from no. 41, same horizon and locality. Suborder Tarsioidea, family Tarsiidæ.

redescribed by Matthew (1915), one pair of the lower front teeth, possibly the opposite canines, is greatly enlarged and procumbent, and produced in rodent-like or diprotodont fashion, the dental formula being $I_0^?$ C_1^1 P_2^2 M_3^3 (Matthew). The enlargement of the lower front teeth has conditioned the loss of the lower incisors, the development of a diastema behind the large procumbent tooth, the deepening of the front part of the mandible, the crowding of the premolar series and the consequent enlargement and strengthening of the insertion

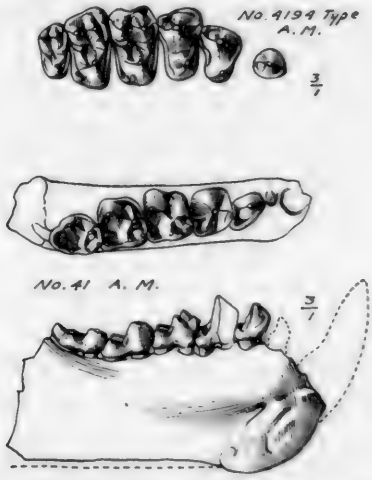

FIG. 119. *Telonius homunculus*. UPPER TEETH AND LOWER JAW, PART OF RIGHT RAMUS. × 3. AFTER MATTHEW

Gray Bull beds (Lower Eocene), Bighorn basin, Wyoming. Suborder Tarsioidea, family Tarsiidæ.

areas of the jaw muscles, both of the skull and mandible. The fourth lower and the fourth upper premolars have large piercing apices, and the first and second lower molars are much widened across the talonids. The upper molars are very wide transversely and narrow anteroposteriorly, with rounded para- and metacones. The third molars are small. These modifications are perhaps adapted either for piercing and breaking the hard bodies of insects or for extracting and crushing seeds from fruits.

FIG. 120. *Tarsius spectrum*

FIG. 121. *Tetonius homunculus*. Amer. Mus., no. 4,194. Gray Bull beds (Lower Eocene), Bighorn basin, Wyoming.

FIGS. 120 TO 123, INCLUSIVE. COMPARATIVE SERIES: SKULLS OF RECENT TARSIUS AND OF EOCENE TETONIUS. × 3

Suborder Tarsioidea, family Tarsiidæ.

FIG. 123. *Tetonius homunculus*

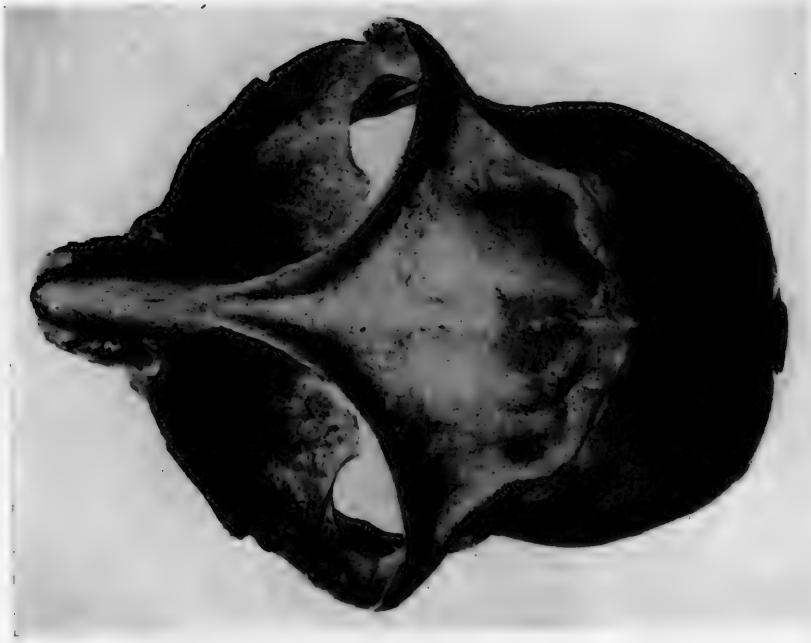

FIG. 122. *Tarsius spectrum*

The orbits (*fig. 123*) are much enlarged, the braincase expanded and the muzzle narrow, so that the general appearance of the skull approaches that of *Tarsius*. Even the construction of the auditory region points in the same direction, since the bullæ were expanded and pressed forward against the lower part of the braincase.

A more detailed examination of the type skull of *Tetonius homunculus* supports the generally accepted view that this animal was a tarsioid, although already too specialized in the dentition to be directly ancestral to the modern *Tarsius*.

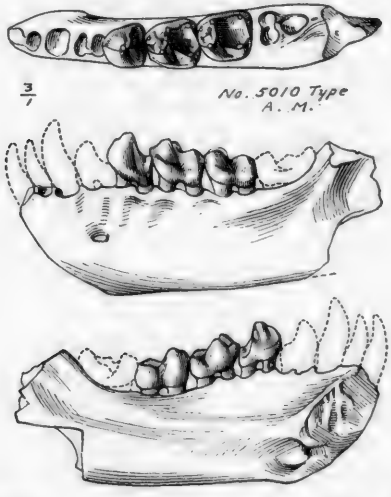

FIG. 124. *Anaptomorphus æmulus*. LOWER JAW (RIGHT RAMUS). × 3. AFTER MATTHEW

Lower Bridger beds (Middle Eocene), Bridger basin, Wyoming. Suborder Tarsioidea, family Tarsiidæ.

Anaptomorphus. This genus (*fig. 124*) has the dental formula $I\frac{2}{2}\ C\frac{1}{1}\ P\frac{2}{2}\ M\frac{3}{3}$, the same as for the Old World series of Primates, but it is a true tarsioid in the form of its lower molars and premolars, and widely different from *Parapithecus*, the oldest and most tarsioid primate of the Old World. Its lower front teeth are not enlarged nor strongly procumbent; the jaw is stout and deep anteriorly. The fourth lower premolar is not as large as in the more specialized genera *Uintanius*

(*fig. 125*) and *Absarokius*. These specializations of the fourth premolars surely remove these genera from ancestral relationships with any of the higher primates.

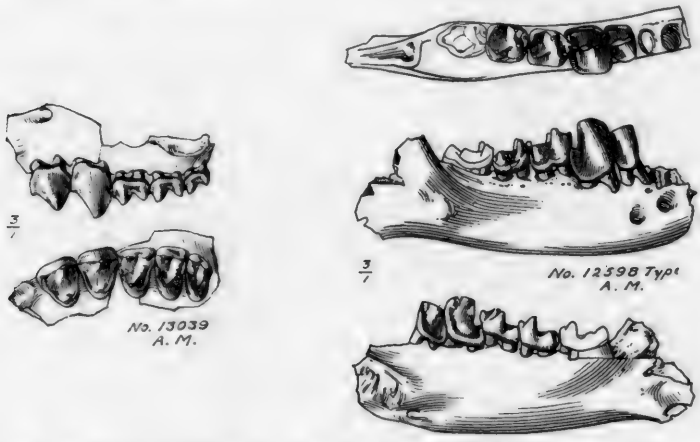

FIG. 125. *Uintanius turriculorum*. LOWER JAW (RIGHT RAMUS) AND LEFT UPPER CHEEK TEETH. × 3. AFTER MATTHEW

Lower jaw, Lower Bridger beds (Middle Eocene), Bridger basin, Wyoming. Upper teeth, Upper Bridger beds (Middle Eocene), Bridger basin, Wyoming. Suborder Tarsioidea, family Tarsiidæ.

EUROPEAN EOCENE TARSIOIDS

In the European Eocene the tarsioid group is represented by seven genera, mostly known from teeth and jaws, except *Pronycticebus* and *Necrolemur*, which are known from well preserved skulls. All have recently been carefully described and figured by Stehlin (1916). The group first appears in the Lower and Middle Lutetian beds, in the second half of the Lower Eocene; and after passing through the Upper Lutetian, Bartonian and Lower Ludian beds, disappears during the Upper Ludian of Upper Eocene age. Its range in Europe is thus nearly contemporaneous with the range of the related genera in America, although not extending so far down into the Lower Eocene as do the latter. No known genera of tarsioids are common to Europe and America; and thus there is evidence that, although derived from

a common stock, perhaps of Lower Eocene age, the tarsioids of the two continents followed independent lines of evolution without faunal interchange (Stehlin). Both the American and European groups are "polyphyletic," and although we know only fragments of different phyla, they afford strong evidence of the evolution of the upper molars from the triangular to the quadrangular stage, in the normal mammalian manner described in earlier parts of this work.

Pseudoloris. Perhaps the most primitive of the European genera is *Pseudoloris* (*figs. 126, 127*), from the Bartonian (Middle Eocene) of France, which has tritubercular upper molars with well developed proto- and metaconules, small cingulum-hypocones and very small protostyles, or projections of the anterointernal cingula. The general form of the upper molars suggests those of the recent lorises and galagos, as noted by Stehlin (1916, p. 1396), but, as he also suggests, the resemblances are not sufficiently close to prove a near relationship, and it is far more probable that *Pseudoloris* is a true tarsioid, only remotely related to the Lorisidæ, which appear to be *Tarsius*-like derivatives of some primitive Eocene lemuroids. *Pseudoloris* is probably related, by descent from a common ancestor, to the American tarsioids. As it had a pair of enlarged lower front teeth, which are regarded by Stehlin as canines, it resembles rather *Tetonius* in this character; on the other hand, its molars recall those of *Omomys*, except for the large size of the metaconules of the upper molars and the loss of the paraconids of the second and third lower molars.

Anchomomys. This genus is known from two species: one in the Lutetian or summit of the Lower Eocene of France and Switzerland, the other in the Bartonian or Middle Eocene of France. It differs from *Pseudoloris* especially in the reduction or absence of the metaconules. In *Anchomomys quercyi* (*fig. 130*) the first and second upper molars are less wide transversely and relatively wider anteroposteriorly than in *Pseudoloris;* the posterointernal corners of the crown are developing, so that the contour is becoming more quadrate. The orbit in this genus was certainly large, as shown by the form of its floor and border, above the maxilla. The mandible of *Anchomomys* cfr. *Gaillardi*, figured by Stehlin (*op. cit.* p. 1412), shows that the dental formula was probably $I\frac{2}{2}\ C\frac{1}{1}\ P\frac{3}{3}\ M\frac{3}{3}$, as in the American *Anaptomorphus*. The two incisors were small and gently proclivous, the canine large and erect.

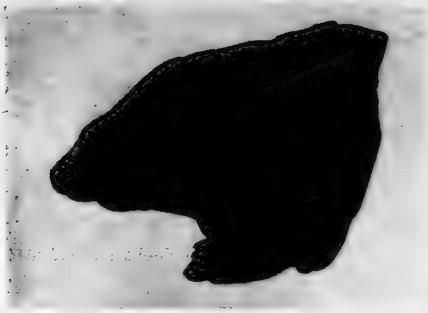

FIG. 126. *Pseudoloris parvulus*. Left maxilla, with p^4-m^3. × 8. Phosphorites near Caylux.

FIG. 128. *Anchomomys gaillardi*. Fragment of left maxilla, with m^1-m^3. From Lissieu near Lyon. × 6.

FIG. 127. *Pseudoloris parvulus*. Left mandible, with p_4-m_3. Phosphorites near Caylux.

FIG. 129. *Anchomomys gaillardi*. Fragment of left mandible, with m_1-m_3. From Lissieu, near Lyon. × 6.

FIGS. 126 TO 129, INCLUSIVE. EUROPEAN EOCENE TARSIOIDS. LOWER AND UPPER CHEEK TEETH. AFTER STEHLIN

Suborder Tarsioidea, family Tarsiidæ.

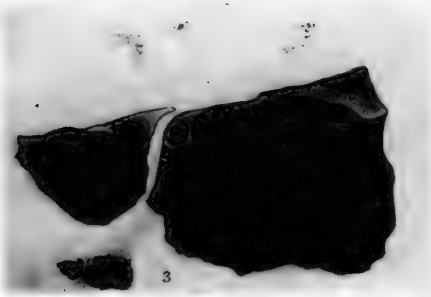

FIG. 131. *Periconodon helveticus*. LEFT MAXILLA, WITH p^3–m^3. × 6. AFTER STEHLIN

From Egerkingen, Switzerland.

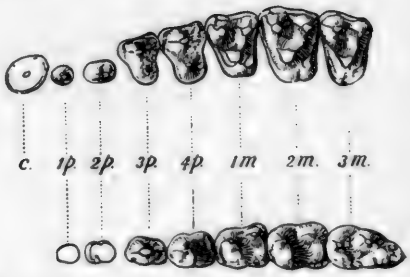

FIG. 132. *Pronycticebus gaudryi*. LEFT UPPER AND RIGHT LOWER CHEEK TEETH. × 2. AFTER GRANDIDIER

From Mermelein (Lot) France.

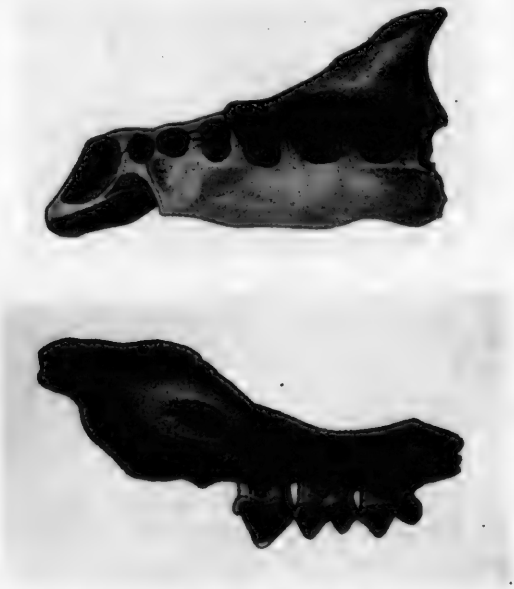

FIG. 130. *Anchomomys quercyi*. FRAGMENT OF LEFT MAXILLA, WITH p^4–m^2. × 6. AFTER STEHLIN

From Phosphorites of Quercy, France.

FIGS. 130 TO 133, INCLUSIVE. EUROPEAN EOCENE TARSIOIDS. UPPER AND LOWER TEETH. AFTER STEHLIN, GRANDIDIER

Suborder Tarsioidea, family Tarsiidæ.

Periconodon. In this more specialized relative of *Anchomomys* from the Lower or Middle Lutetian of Switzerland, the metaconules of the upper molars (*fig. 131*) have been suppressed and the cingulum-hypocones and protostyles have become quite prominent. Stehlin (*op. cit.*, p. 1432) notes the striking resemblances of these teeth to the molars of the recent South American monkeys *Chrysothrix* and

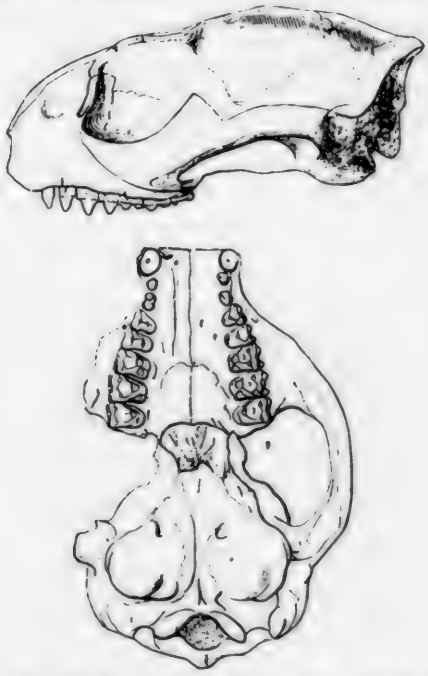

FIG. 133. *Pronycticebus gaudryi.* SKULL, × 1. AFTER GRANDIDIER

Phosphorites (Middle Eocene), of Mermelein (Lot), France. Suborder Tarsioidea, family Tarsiidæ (?).

Cebus, but concludes that the hiatus in geological time between the recent and the Eocene primates is far too great to permit us to regard the resemblance in question as indicating close relationship.

Pronycticebus. This animal was regarded by its discoverer, Grandidier (1905), as a forerunner of the modern loris, but Stehlin has shown (1916 p. 1422) that its dentition (*figs. 132, 133*) rather indi-

cates relationship with *Anchomomys*. The upper canine was relatively large with a rounded rather than compressed base. All four premolars are retained in both the upper and lower jaws. The first upper premolar is very small and peglike; the second, larger and slightly more advanced but widely different from the third premolar, which is approaching the condition of p^4. The latter is bicuspid and quite different in form from m^1. The first and second upper molars are of primitive tritubercular type with V-shaped cusps, well developed external cingula, complete proto- and metaconules, and hypocones connected with the cingulum. The third molar is tritubercular with a well developed internal cingulum not rising into a distinct hypocone. The lower cheek teeth are correspondingly primitive and approach the common plan of the primitive Lemuroidea. The skull is also more primitive, more like that of the primitive Notharctidæ, than is that of any other known tarsioid, the face being less reduced than in the known tarsioids, while the braincase, orbits and auditory bullæ are less expanded.

Nannopithex. The curious type called *Nannopithex polycarus* by Stehlin (*op. cit.* p. 1392) is represented by a fragment of the right maxilla containing three upper molars (*fig. 134*). The tritubercular first and second upper molars recall those of the American *Hemiacodon* in the unusually large size of the circular metaconules and in the strong development of the cingulum-hypocone. On the other hand, they foreshadow the upper molars of *Necrolemur* cfr. *Zitteli* (*fig. 135*) in the peculiar relations of the protoconule fossa to the anterior slope of the paracone and in the appearance of the large, conspicuous, conical metaconule. The most striking difference is that the cingulum-hypocone is distinct from the posterior slope of the protocone, whereas in *Necrolemur* it is connected with it. However, in the third molar (m^3) the cingulum lacks a distinct hypocone and is connected with the posterior slope of the protocone, as in *N. zitteli*. *Nannopithex* also lacks the small accessory cusp between the metaconule and the protocone which is incipient in some specimens of *N. zitteli*. Nevertheless the anterior slope of the protoconule of *Nannopithex* is conspicuous and could readily give rise to the conditions in *Necrolemur*.

Stehlin has noted these and other resemblances, but refers to them only as analogies (p. 1393) and doubts the relationship (p. 1395), as

he ascribes great importance to the differences in the postero-internal cusp; but he suggests a possible relationship between *Nannopithex* and the American *Washakius*.

But, even if *Nannopithex* be not directly ancestral to *Necrolemur*, it illustrates a stage of molar evolution tending to connect the highly

FIG. 134. *Nannopithex pollicaris*. m^{1-3} RIGHT. × 8

From the lower Eocene of Egerkingen, Switzerland. Suborder Tarsioidea, family (?) Microchœridæ.

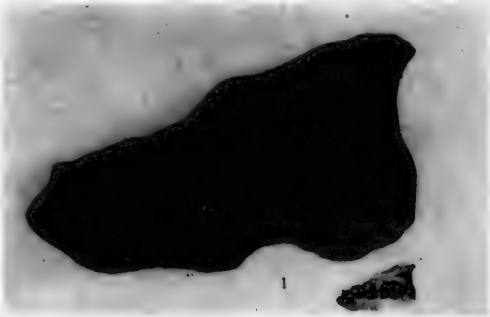

FIG. 135. *Necrolemur* CFR. *Zitteli*. LEFT MAXILLA, WITH p^3–m^3. × 4

From the Lower Eocene of Egerkingen, Switzerland. Suborder Tarsioidea, family Microchœridæ

FIGS. 134, 135. COMPARISON OF NANNOPITHEX POLLICARIS AND NECROLEMUR CFR. ZITTELI. AFTER STEHLIN

specialized conditions of the upper molars of *Necrolemur* with the more primitive tarsioid stage preserved in *Hemiacodon* and *Washakius*.

Necrolemur and Microchœrus. *Necrolemur antiquus* (*fig. 136*) is characterized by the elaborate pattern of the upper molar crowns in

which there was a strong tendency toward the development of extra conules and the division of the upper molar crown into anterior and posterior moieties.

According to Stehlin (*op. cit.*, p. 1329) the dental formula was $I\frac{2}{0}\ C\frac{1}{1}\ P\frac{3}{4}\ M\frac{3}{3}$. There was a pair of enlarged proclivous lower front teeth (*fig. 137*), which Stehlin regards as canines. This specialization, with others, removes *Necrolemur* from the line of ascent either to *Tarsius* or to any of the higher primates.

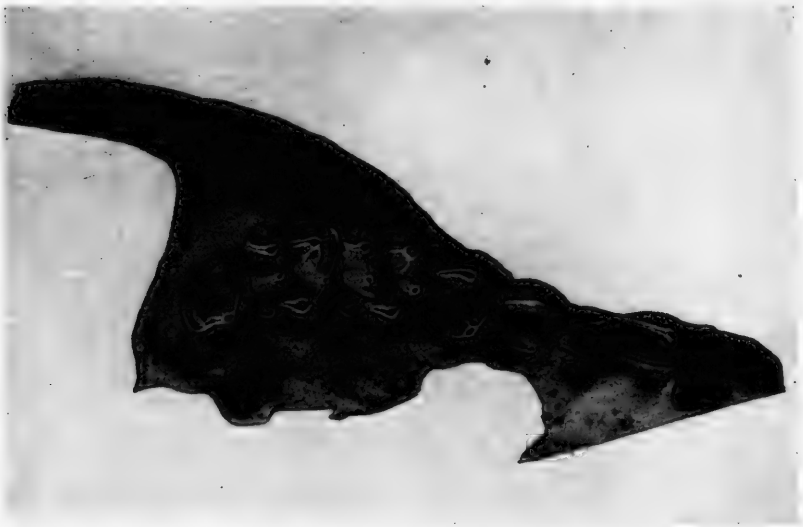

FIG. 136. *Necrolemur antiquus*. RIGHT MAXILLA, WITH LATERAL INCISOR, CANINE AND CHEEK TEETH. × 5. AFTER STEHLIN

From the Phosphorites of Largnol, France. Suborder Tarsioidea, family Microchœridæ.

The skull of *Necrolemur* (*figs. 137, 138, 139, 141*), as known from beautifully preserved specimens, is of a highly modified tarsioid type, with convergent upper dental arches and excessive enlargement of the auditory bullæ, which press forward into the braincase in front of them and are provided with bony external auditory tubes, as in *Tarsius*. The orbits are much enlarged, the interorbital portion of the

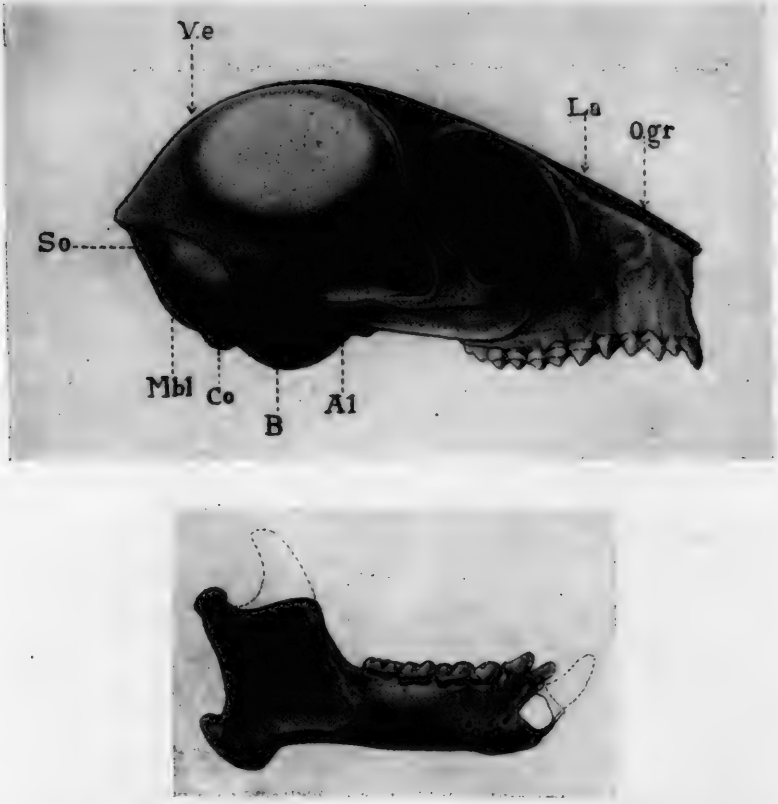

FIG. 137. *Necrolemur antiquus*. RECONSTRUCTION OF SKULL AND LOWER JAW. × 2. AFTER STEHLIN.

From the Phosphorites (Middle Eocene) of France. Suborder Tarsioidea, family Microchœridæ.

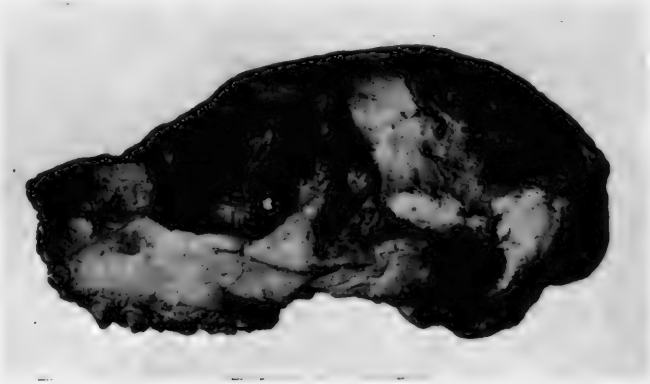

FIG. 138. *Necrolemur antiquus*. SKULL IN THE PRINCETON UNIVERSITY PALÆONTOLOGICAL COLLECTION. × 2

Courtesy of Dr. W. J. Sinclair.

braincase is reduced to a thin septum; the construction of the lacrymal region is also of tarsioid type. On the other hand, the great inflation of the mastoid region heightens the general resemblance to the skull of *Galago* (*fig. 140*).

The tendency toward a multiplication of small cusps on the surface of the upper molar crowns culminates in the *Microchœrus ornatus*

FIG. 139. FIG. 140.

FIGS. 139, 140. COMPARATIVE SERIES: SKULLS OF NECROLEMUR ANTIQUUS AND GALAGO CRASSICAUDATUS. × 2

Specimens in the Peabody Museum of Comparative Zoölogy, Harvard University. Courtesy of Dr. Samuel Henshaw. Illustrating the close convergence in skull form between these two genera, which belong respectively in the suborders Tarsioidea and Lemuroidea.

of Stehlin, in which the molar crown of m^2 (*fig. 142*) is surmounted by no less than eleven cusps and cuspules. Both the proto- and the metaconule appear to be double. There is a large mesostyle and an accessory stylar cusp on the cingulum-hypocone. The contours of the

FIG. 141. *Necrolemur antiquus*. PALATAL VIEW OF SKULL. × 3

Specimen in the Museum of Comparative Zoölogy, Harvard University. Courtesy of Dr. Samuel Henshaw. Suborder Tarsioidea, family Microchœridæ.

upper molars are subquadrate and the interdental spaces are practically obliterated. This pseudo-multituberculate specialization evidently preceded the extinction of the family.

The differences separating *Necrolemur* and *Microchœrus* from any of the New World or Old World monkeys are numerous and important, and there is no evidence for deriving any of the higher types directly

FIG. 142. *Microchœrus ornatus*. RIGHT MAXILLA, WITH p^2–m^3. × 6. AFTER STEHLIN

Upper Eocene of France.

FIG. 143. *Microchœrus ornatus*. RIGHT LOWER CHEEK TEETH, p_3–m_3, THE FIRST TOOTH DISPLACED. × 4. AFTER STEHLIN

Suborder Tarsioidea, family Microchœridæ.

from this source. Nevertheless these genera exhibit certain important advances in the direction of the Old World primates, among which may be noted the development of quadrate upper molars with subequal anterior and posterior moieties, the development of hypoconulids in the lower molars (*fig. 143*), the final loss of the paraconids in the lower molars and the tendency for the reduction of the trigonid basins, the tendency for p^4 and p^3, and the corresponding lower teeth, to

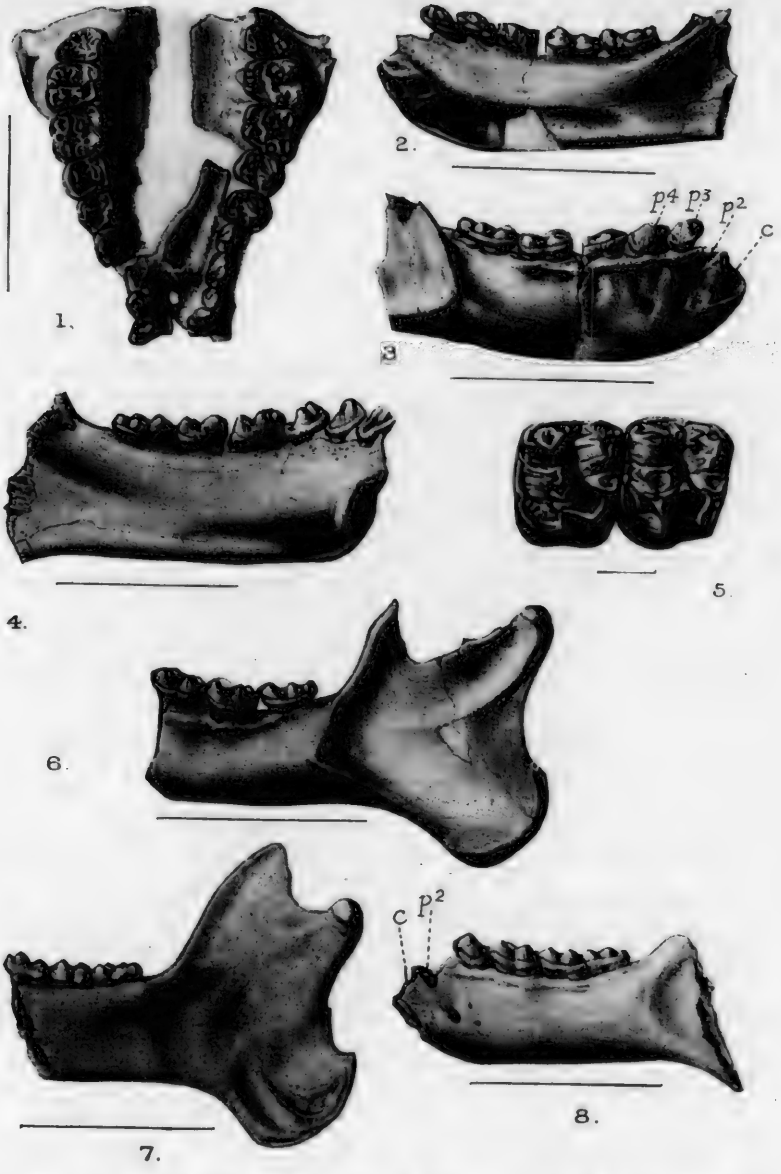

FIG. 144. *Microchœrus* AND *Necrolemur*. ENLARGED. AFTER FORSTER-COOPER

1.—*Microchœrus erinaceus*. Palate. British Museum. Upper Eocene, Hardwell, Isle of Wight.

2.—Ditto. Right lower ramus, inner side (Cambridge).

3.—Ditto. Right lower ramus, outer side (British Museum). The small socket for the second tooth can be made out as a small black dot at the upper point of the large socket.

4.—Ditto. Right lower ramus, inner side (Cambridge).

5.—Ditto. Upper molars (British Museum).

6.—Ditto. Hind part of left ramus (Cambridge).

7.—*Necrolemur edwardsii*. Hind part of left ramus (British Museum).

8.—Ditto. Front part of left ramus (British Museum).

The small black lines show the actual sizes of the specimens.

become bicuspid, the incipient development of a bony postorbital partition, the lateral expansion of the base of the braincase, the development of a tubular external auditory meatus, and the tendency for the angle of the mandible to be expanded and for its posterior border to become very large and rounded. Thus *Necrolemur* and *Microchœrus* have advanced far from the primitive tarsioid type and indicate some of the structural stages through which the actual ancestors of the Old World series probably passed.

THE MODERN TARSIUS

The systematic position and relationships of the Spectral Tarsier, of Borneo and other Malayan islands, including the Philippines, have constituted a long debated question that has a direct bearing upon the problem of man's ancestry. As noted in Part II of this work, *Tarsius* was formerly classed among the Lemuroidea, but all recent investigation has tended to confirm its right to be assigned, together with its Eocene relatives, to a distinct suborder Tarsioidea, coördinate with the other two suborders, Lemuroidea and Anthropoidea. Neither *Tarsius* itself nor its known Eocene relatives, described above, appear to be directly ancestral either to the platyrrhine or to the catarrhine divisions of the Anthropoidea. Nevertheless *Tarsius* parallels the higher primates in so many characters of the brain, skull, reproductive organs and other parts, that a very remote common ancestry of the three suborders seems highly probable.[2] Moreover the earliest known and by far the most primitive representatives of the Anthropoidea, namely, the genus *Parapithecus* from the Lower Oligocene of Egypt, carries that group well back toward a short-jawed and probably large-brained stem, which might be described as tarsioid in a broad sense; while, as will presently be shown, the various Platyrrhinæ appear also to have been derived from another short-jawed and large-brained stock, belonging perhaps to a quite different family of the ancestral tarsioid primates.

With regard to the derivation of *Tarsius*, very possibly *Anchomomys* was more or less closely related to its Eocene ancestors, but

[2] In this connection see the recent "Discussion on the zoological position and affinities of *Tarsius*:" *Proceedings of the Zoölogical Society, of London*, 1920 (Feb.), p. 465.

the loss of the paraconids in the lower molars (*fig. 127*) appears to remove *Anchomomys* from the direct line of ascent (Stehlin, p. 1426). The modern genus, although retaining much of its Eocene heritage, has reduced metaconules in the upper molars (*fig. 120*), and it has become further specialized in the extreme enlargement of the orbits (*fig.*

FIG. 145. THE SPECTRAL TARSIER (*Tarsius spectrum*). AFTER DUCKWORTH
Suborder Tarsioidea, family Tarsiidæ.

122). Its front teeth are moderately specialized by the reduction of the lower incisors to one on each side. This nocturnal and arboricolous animal is said to feed largely upon insects and small reptiles (Lydekker).

The palæontological evidence reviewed in this work is surely against Wood-Jones's view that the existing *Tarsius* is the nearest living relative of man. *Tarsius* may well parallel the human condition in the

construction of the placenta, and in a few other points noted by Wood-Jones (to all of which I have given careful consideration), but its relationships to man are plainly very indirect and must be traced backward along gradually converging lines to the primitive tarsioid stocks which gave rise at different times and at different places to the higher groups of primates.

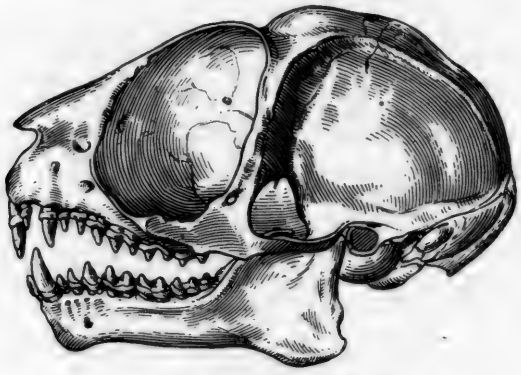

FIG. 146. *Tarsius spectrum*. SKULL. × 2

Suborder Tarsioidea, family Tarsiidæ.

IV. THE SOUTH AMERICAN MONKEYS (PLATYRRHINAE)

TERTIARY FOSSIL PLATYRRHINÆ

Of the few South American Tertiary genera which have been attributed to this group, only one of them, the *Homunculus patagonicus* (*fig. 147*) of Ameghino, from the Santa Cruz Miocene of Patagonia. is known from a well preserved jaw and the fore part of the skull. Although this genus was already a true platyrrhine monkey, it does carry us back a short way toward the stem of the group. According to Bluntschli (1913), who has carefully studied the original specimens, the closest resemblances of *Homunculus* are with the smaller Cebidæ, especially "*Nyctipithecus*" (*Aotus*) and "*Callithrix*" (*Callicebus*); genera which, for reasons set forth below, appear to have retained many primitive or stem characters.

Another Santa Cruzian form, *Anthropops perfectus* (*fig. 148*) of Ameghino, based on the front end of a lower jaw, has a deep chin that is even better developed than that of *Cebus*. Its dental formula was $I_2\ C_1\ P_3\ M_?$; the first two premolars (p_2, p_3) were small. Bluntschli states that very probably "*Anthropops*" is identical with *Homunculus*. Still another Santa Cruz type, referred to the primates by

FIG. 147. *Homunculus patagonicus*. SKULL AND JAW. × 1. AFTER F. AMEGHINO

Santa Cruz (Lower Miocene), Patagonia. Suborder Anthropoidea, section Platyrrhinæ, family Cebidæ.

Ameghino and by Abel, is *Eudiastatus lingulatus* Ameghino (*fig. 148*), based on the front end of a small jaw; this bears on the inner and ventral side of the symphysis a very large median tubercle in place of the usual paired pits for the tendons of the geniohyoid muscles. A faint suggestion of this condition may be seen in some jaws of *Cebus* and *Alouatta*. The dentition is very poorly preserved. According to Bluntschli (*op. cit.* p. 38) *Eudiastatus* is not a primate at all but may be a bat.

Neither the "chin" of *Anthropops* nor the "genial tubercle" of *Eudiastatus* afford the least evidence of special relationship with Man.

It is therefore apparent that the direct palæontological record of the evolution of the Platyrrhinæ does not extend below the Miocene, and we are therefore limited to indirect evidence as to the earlier course of evolution and derivation of the group.

FIG. 148. *Anthropops perfectus* (A) AND *Eudiastatus lingulatus* (B). LOWER JAW FRAGMENTS. × 1. AFTER F. AMEGHINO

Santa Cruz (Lower Miocene), Patagonia.

Bluntschli (1913, p. 38), who has examined these remains, states that the lower jaw of "*Anthropops*" probably belongs to *Homunculus*, while "*Eudiastatus*" is not a primate at all, but probably a bat.

WERE THE PLATYRRHINÆ DERIVED FROM NOTHARCTUS?

In 1904 Wortman's investigations led him to conclude that while the American Eocene tarsioids were related to the modern *Tarsius*, they were also related to the ancestors of the higher primates, so that he transferred all the known tarsioids under the group name Paleopithecini to the suborder Anthropoidea. He also suggested that *Omomys* and *Washakius* (described above), "as far at least as we are permitted to judge from their scant remains, are closely related to *Adapis* and *Notharctus*, but had made greater progress in the reduction of the premolars. This gives an especially monkey-like appearance, pointing particularly in the direction of certain living Cebidæ." He also concluded that "it is in just such a group as that which includes *Adapis*, *Notharctus* and *Limnotherium*, that we must seek for the beginnings of the higher monkeys and apes which follow; and while these species may not have been in the direct line of descent, they

cannot at the same time have been far removed from it (1904, pp. 172, 173)."

This concept unquestionably overemphasizes the nearness of the tarsioids to the Adapidæ. At least as far back as the Lower Eocene the tarsioids and the Adapidæ were wholly distinct families. As I have elsewhere shown (1920), the Adapidæ were still in a primitive lemuroid stage of evolution in the great majority of their cranial, dental and skeletal characters, while the tarsioids (as Wortman himself recognized) were precociously specialized in many respects. Nevertheless it is possible that Wortman may have been right in his general conclusion, and that the platyrrhine series have been derived from the Notharctidæ. This subject has a bearing upon the origin of man and the evolution of the human dentition, and it therefore calls for fairly detailed consideration at this point.

If the South American monkeys have been derived from large and progressive Notharctidæ, such as *Notharctus crassus* (see Part II), then very probably the most primitive dentition of the series is that of the Howler monkeys, *Mycetes* (= *Alouatta*). Unfortunately, however, we have but little means for judging whether the resemblance in the cheek teeth (*fig. 149*), which is by no means close, is a direct heritage from the later Notharctidæ, or whether it is due largely to homoplastic evolution. *Alouatta* (*fig. 151*) differs from *Notharctus* (*fig. 150*) in the marked shortening of the bony face, and it is in every important character a true platyrrhine monkey. The resemblances to *Notharctus crassus* are accompanied by an obviously high degree of specialization of the basihyal bone, which is of enormous size and greatly inflated. The lower jaw is also deepened and expanded in such a way as to provide sufficient room in the throat for this great resonating pouch. This expansion of the jaw has not only provided opportunity for great increase in the jaw muscles, but is also accompanied by a very pronounced upward tilting of the braincase.

Possibly the curious characters of the lower jaw and skull may have been partly developed before the great expansion of the resonating pouch in the throat, because they are partly foreshadowed in the genera *Callicebus*, *Aotus* (*Nyctipithecus*), *Brachyteles* (*pl. 8–9*). These forms are also the ones in which the molar patterns appear to

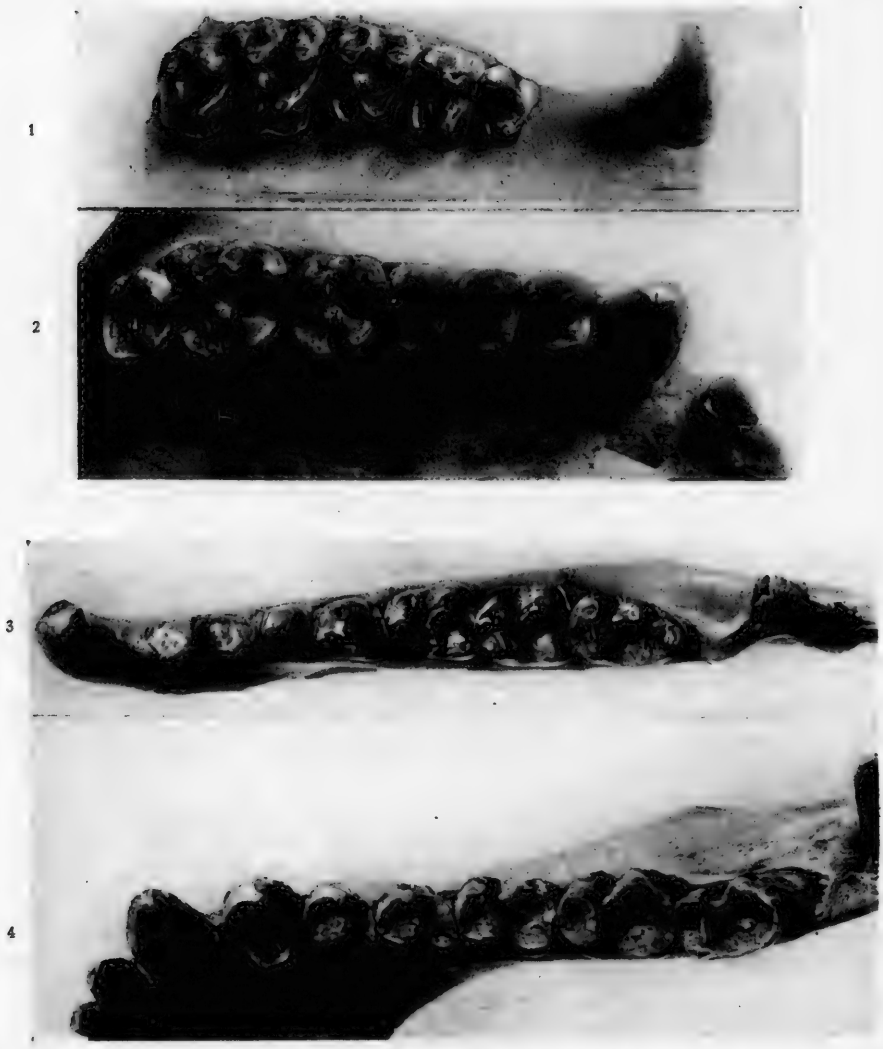

FIG. 149. COMPARISON OF TEETH OF AMERICAN EOCENE PRIMATE *Notharctus* AND RECENT PLATYRRHINE MONKEY *Alouatta*

1.—*Notharctus crassus*. Right upper cheek teeth (p^1, p^2 not preserved). × 2. Amer. Mus. no. 11,689. Upper Bridger beds (Middle Eocene), Wyoming. Suborder Lemuroidea, family Notharctidæ.

2.—*Alouatta* sp. Howler Monkey. Right upper jaw. × 2. Recent, South America. Suborder Anthropoidea, family Cebidæ.

3.—*Notharctus crassus*. Right lower jaw. × 2. Amer. Mus. no. 11,689. Upper Bridger beds (Middle Eocene), Wyoming. × 2.

4.—*Alouatta* sp. Amer. Mus. no. 14,660. × 2.

FIG. 150. *Notharctus osborni*. × 1. Amer. Mus. no. 11,466. Lower Bridger beds (Middle Eocene), Bridger basin, Wyoming.

FIG. 151. *Alouatta beelzebul*. × 3/4

FIGS. 150, 151. COMPARISONS OF SKULLS OF EOCENE LEMUROID *Notharctus* AND MODERN PLATYRRHINE MONKEY, *Alouatta*

be fairly primitive, and *Callicebus* seems to be on the whole the most primitive and also the most "tarsioid" (in a broad sense) of the entire platyrrhine series. In this genus the molars (*pl. 11*) are of comparatively primitive tritubercular type with distinct remnants of the V-shaped form of the para- and metacones, and sharp crests on the anterior slopes of the protocones. The hypocones are connected with the posterior slopes of the protocones and are therefore apparently pseudohypocones like those of the Notharctidæ. This is especially evident in the specimen of this genus figured by Winge (1895). In *Aotus* (*Nyctipithecus*) on the other hand, the hypocones are quite prominent and appear to be connected with the cingulum (*pl. 11*). In *Alouatta* (*pl. 11*) they are connected both with the cingulum and with the crest of the protocone; in many or perhaps all of the remaining Cebidæ (*pl. 11*) and Hapalidæ (*pl. 13*), the hypocones are widely connected with the cingula and well separated from the protocones by a deep cleft. Thus the more primitive genera approach the conditions in the Notharctidæ, while the more specialized have lost them. But instead of postulating a direct derivation of the modern *Alouatta* from the Eocene *Notharctus crassus*, it seems safer to regard the Notharctus-like characters of the *Alouatta* molars as partly homoplastic and secondary, partly because they are associated with so many obviously peculiar specializations in the rest of the skull and skeleton, partly because they are adapted to the leaf-eating habits of the animal—an exceptional diet for a member of the Platyrrhinæ, which feed typically on fruits and insects.

For the present, therefore, the available evidence indicates that the stem Platyrrhinæ had many characters of the Notharctidæ, together with others like those of certain Eocene tarsioids. The auditory region of the skull (*plates 11, 13*), which usually furnishes important evidence as to the relationships of groups, points in the same direction, that is, the conditions in the Platyrrhinæ are far more advanced than those in the Notharctidæ, but might be derived eventually from them.[3]

Unfortunately in the absence of annectant forms from intermediate horizons, we cannot assert that the Notharctidæ are the direct

[3] A more detailed discussion of this subject is given in my memoir on Notharctus (1920, p. 220).

ancestors as well as the remote structural ancestors of the Platyrrhinæ. The American Eocene genus *Aphanolemur* (*fig. 152*) of Granger and Gregory (1915) has a more expanded braincase and a shorter face than the typical Notharctidæ, and possibly it is sufficiently like the tarsioids to be a direct ancestor of the Platyrrhinæ.

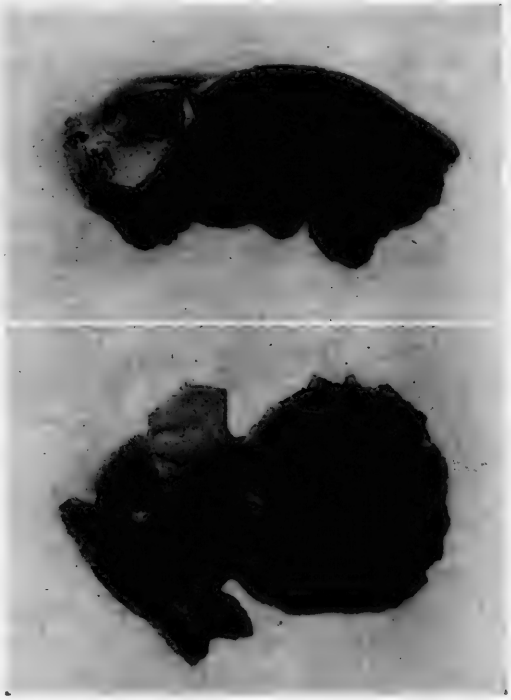

FIG. 152. *Aphanolemur gibbosus*. IMPERFECT SKULL. × 1. AFTER GRANGER AND GREGORY

Yale University Museum. Lower Bridger beds (Middle Eocene). Suborder Lemuroidea, family uncertain.

Of considerable importance are the characters of the incisors and canines, which in the South American series (*plates 8, 9, 11, 12, 13*) avoid all the peculiar specializations observed in the lemuroids and in most known tarsioids, and are closely comparable with the conditions in the Notharctidæ described in Part II of this review. The

diverse specializations of the front teeth in these other families are such as to remove most of the known genera definitely from direct ancestry to the Platyrrhinæ, and therefore they serve to enhance the value of the relatively close agreement in this region between the platyrrhines and the Notharctidæ.

ADAPTIVE RADIATION OF THE CEBIDÆ

The existing South American monkeys exhibit a considerable adaptive radiation (*plates 9–13*) in the dentition and skull, but in view of the poverty of palæontological evidence it was for a long time difficult to decide which are the more primitive species. After repeated comparisons of the skulls and dentitions of the recent platyrrhine genera, during several years past, I have now adopted the provisional conclusion that in the construction of the skull and in the characters of the dentition the genera *Callicebus* and *Aotus* ("*Nyctipithecus*") are on the whole the most primitive and "tarsioid" living cebids, while *Cebus* and *Chrysothrix* (*Saimiri*) are the most advanced and pithecoid.

From or near *Callicebus* (*pl. 8*) as a starting point we may trace "an adaptive radiation" in skulls and dentitions, one morphological series leading through *Brachyteles*, *Ateles* and *Homunculus* to *Lagothrix*, *Cebus* and *Saimiri*. In this line the back part of the jaw becomes shallower, the upper and lower canines increase in size, and a bony chin and wide intercanine diameters are finally developed. Meanwhile the tooth rows (*plates 11, 13*) become straight or slightly divergent and the molars vary from the subtriangular to the subbilophodont condition. The anterior premolars, at first small, finally become quite large (*Cebus*); the face (*plates 8, 9*) becomes shallower and more protruded below the orbits; and the zygomatic arches, originally stout and inclined downward, become slender and horizontal; the cranium (*plate 10*) finally bulges greatly behind the foramen magnum, and is very dolichocephalic; the outer borders of the orbits meanwhile grow forward so that the eyes finally look more directly forward instead of partly outward, and they have also become separated from the temporal fossæ by bony partitions.

More in detail, the steps by which the platyrrhine skull became modernized from a tarsioid to a pithecoid stage may be visualized as follows: First, in correlation with the rapid expansion of the brain

there has been a steady and moderate increase in the breadth of the braincase across the parietal, a rapid increase in the frontal region just behind the orbit, accompanied by a rapid increase in antero-posterior diameter, so that in the higher Platyrrhinæ (*Cebus*, *Chrysothrix*) the general form of the cranium as seen from above finally becomes like a stout, wide-necked vase (*pl. 10*). Secondly, the outer rim of the orbits has grown forward, while the inner borders have been brought nearer to the midline, greatly restricting the interorbital, nasal and lacrymal regions, the end result being that the axis of the eyes point forward rather than sideways. This has been associated with a shortening and a retraction of the face beneath the orbit, and with a progressive separation of the orbits from the temporal fossæ by bony partitions developed chiefly on the frontals and malars, and partly on the sphenoid. Possibly the first development of this partition may have been in response to an enlargement of the orbits connected with nocturnal habits. Indeed the available evidence suggests that in many phyla of Platyrrhinæ, such as the marmosets and the squirrel monkeys, the relatively small size of the orbits is secondary.

The skull of *Aotus* (*Nyctipithecus*) is near to the primitive *Callicebus*-like type, differing in the large size of the orbits (*plates 8, 10*). The orbito-temporal fissure (*pl. 11*) is unusually large in this form, which may indicate that the incomplete condition of the postorbital septum is a primitive inheritance from remote tarsioid ancestors. The short, wide cranium (*pl. 10*) seems much more primitive than the excessively elongate cranium of the squirrel monkeys.

The early shortening of the face and the transverse expansion of the braincase, which increased the intercondylar diameter of the jaw, together with the relatively small size of the incisors and canines, were at first concomitant with a marked convergence of the opposite dental arches, which were more or less pointed in front, both in the upper and lower jaws. These primitive conditions are largely retained in the existing *Callicebus*, but in the more specialized genera the upper canines become enlarged and the inter-canine diameter much increased, so that the canines protrude at the side and the rows of opposite cheek teeth become parallel or even slightly divergent anteriorly. Meanwhile the lower jaw, at first deep posteriorly and shallow

anteriorly, becomes wider and flatter across the symphyses; a bony chin is finally developed and the center of power moves forward from the back part of the jaw, the angular region becoming smaller and the vertical depth of the body of the jaw decreasing.

The shortening of the bony face perhaps conditioned the crowding out of the first premolar, both in the upper and lower jaws, but the remaining three premolars stoutly asserted themselves against further encroachment, and indeed in several cases, *e.g.*, *Cebus*, *Lagothrix*, and *Saimiri* (*pl. 11*), became larger than the molars.

In the Notharctidæ, which in general are far older and more primitive than the Platyrrhinæ, there was a progressive development of the premolars as we pass from p^1 to p^4; but in the typical Platyrrhinæ all three premolars tend to become bicuspid with oval crowns and low rounded cusps. It is chiefly in *Brachyteles*, *Alouatta* and *Callicebus* that vestiges of more primitive conditions may be observed. Perhaps the most extreme advancement in the premolars is seen in *Cebus*, in which the anterior upper premolars have shared in the enlargement of the canines and are wide, completely bicuspid teeth (*pl. 11*). This bicuspid form of the upper premolars has probably contributed to the erroneous idea of some authors that the Hominidæ have been derived from some platyrrhine with an expanded braincase. In *Alouatta* the fourth lower premolar (*fig. 149*) especially shows vestiges of the conditions seen in *Notharctus crassus* in the form and arrangement of the cusps both of the trigonid and of the talonid.

In general the Cebidæ feed upon a mixed diet of fruit and insects; except *Alouatta*, which is said to limit its diet to leaves. Probably if we had fuller information we should find a closer correlation between the particular kinds of insects fed upon by the different genera and the detailed characters of the dentition.

Besides the main morphological series of skulls and dentitions described above, there are several aberrant side branches. First that of *Alouatta*, which may be derived from that of *Callicebus* by a great increase in size, inflation of the larynx, consequent upturning of the cranium posteriorly and marked anteroposterior expansion of the first and second upper molars (*fig. 149*), involving the development of mesostyles and V-shaped para- and metacones. A parallel adaptation to leaf-eating habits occurs in the Indrisidæ.

If, as seems improbable, the V-shaped cusps and well developed mesostyles of the *Alouatta* molars were primitive, that is, by direct derivation from the conditions in *Notharctus crassus*, then the lack of a mesostyle and the low condition of the hypocone in the upper molars of the other genera would be secondary. However, the antero-posterior expansion of the molars in *Alouatta* may rather be dependent upon the greatly increased size of the animal, while the expansion of the whole jaw region may have afforded opportunity for the exceptional development of the hypocones and of the mesostyles, and thus have brought about a secondary resemblance to the molar pattern of *Notharctus crassus* (*fig. 149*). It has already been noted that, as *Alouatta* is only a single and probably specialized type of this family, it is safer to assume that the more tritubercular molars of *Callicebus* (*pl. 11*) are relatively primitive and that the line of molar evolution passes through stages represented in *Callicebus* and *Brachyteles* toward that of *Alouatta*. The other Cebidæ depart further and further from the primitive molar patterns of *Callicebus*. In some of them (e.g., *Cebus*) the first and second upper molars tend to become divided into anterior and posterior moieties through the slight antero-posterior widening of the upper molars and the marked separation of the hypocones. Thus an incipiently bilophodont condition may be produced, which may be a progressive adaptation to insectivorous and frugivorous habits. In *Cacajao* and *Pithecia* the molar cusps tend to lose their sharp relief and distinctness (*pl. 11*). The upper molar crowns become concave transversely and a somewhat squirrel-like molar pattern is produced. This is associated with a marked grinding action of the mandible, and with the development of semi-procumbent upper and lower incisors. Thus these genera have departed very widely from the primitive conditions, and there is no doubt that the observed conditions are secondary and not primitive platyrrhine characters.

The third upper molars of the Platyrrhinæ are relatively small, sometimes dwindling greatly, as in *Saimiri*, *Cebus* and *Aotus*, and foreshadowing the loss of these teeth in the Hapalidæ (*pl. 13*). But this reduction of the third molar was already foreshadowed in the Notharctidæ and is in fact a primitive primate character.

Rather primitive lower molars are preserved in *Alouatta* (*fig. 149*), while fundamentally similar lower molar patterns were present in the Miocene genus *Homunculus* (*fig. 147*). In *Alouatta* the trigonids are distinctly primitive, lacking only the paraconids of the *Notharctus* molars. The hypoconids are larger than in the Notharctidæ and the entoconids have become well defined. The talonid basin is moderately expanded to receive the tip of the large protocones of the upper molars, but there is no excessive transverse widening of the talonid in any of the Platyrrhinæ such as occurs in many tarsioids. In the more advanced platyrrhines (e.g., *Cebus*) the paraconids are lost, and the posterior wall of the trigonid connecting the protoconid and the metaconid is near the anterior border of the tooth, while the talonid fossa is expanded. This parallels the conditions observed in the Old World monkeys and is associated with the obliteration of the interdental spaces through the filling out of the hypocones. The hypoconids are often connected with the entoconids by a low narrow crest homologous with the posterior limb of the talonid-V of Notharctus. The third lower molars of *Alouatta* (*fig. 149*) and of all the other Cebidæ have lost the hypoconulids, but are readily derivable from the primitive notharctid type.

STEM CHARACTERS AND ORIGIN OF THE PLATYRRHINÆ

We are now in a position where we can reconstruct provisionally the stem characters of the platyrrhine skull and dentition. I conceive the stem Platyrrhinæ as small primates in a somewhat tarsioid stage of skull evolution, having the following characters: dental formula, $I\frac{2}{2}\ C\frac{1}{1}\ P\frac{3}{3}\ M\frac{3}{3}$; face short; braincase expanded; orbits relatively small, well-rimmed, directed partly forward; lacrymal mostly within orbits; nose wide, flat; auditory bullæ expanded; tympanic bones large, ring-like; front teeth not rodent-like, but "normal;" dental arches pointed; lower jaw short, deep; canines small, no bony chin; zygomatic arches stout, pitching sharply downward in front; all three upper premolars bicuspid; upper molars tritubercular, with small hypocones and reduced proto- and metaconules; lower molars with trigonid but little elevated above talonid, the latter large but not excessively widened, paraconids reduced or absent, hypoconulids present.

These characters, which are largely retained in some of the more primitive existing Cebidæ, as a whole exclude the Platyrrhinæ from ancestry to the Old World series; so that the two series appear to be parallel and independent branches from a more primitive and more tarsioid ancestral stock.

When and where these hypothetical ancestral tarsioids lived are questions outside the scope of the present inquiry. Those who believe in a lost mid-Atlantic land-bridge, or archipelago, would doubtless put them there, in Upper Cretaceous times, along with many other lost mammalian types that are supposed to have spread eastward and westward into the palæontologically known Eocene regions of Europe and America. The existence of the hypothetical mid-Atlantic land, at least during the Tertiary, is denied, however, on strong palæontological evidence by Dr. W. D. Matthew (1904), who holds that the Paleocene and Eocene faunas of Europe and North America were derived from older centers of dispersal (probably Upper Cretaceous) in the northern circumpolar land mass.

One may therefore assume as a working hypothesis that in this Upper Cretaceous northern hemisphere (the climate of which is known to have been temperate) there was a widely distributed and considerably differentiated pro-tarsioid group, derived eventually from more primitive lemuroids of the Paleocene or of the Upper Cretaceous, and that different families of the pro-tarsioid group spread southward to give rise first to the known tarsioids of Eocene Europe and North America; and somewhat later and at different times, to both the New World and the Old World series.

THE MARMOSETS (HAPALIDÆ)

The taxonomic position and relationships of the Hapalidæ, or marmosets, have in several ways an important bearing on the problem of the origin of man, first, because certain observers are inclined to regard these little animals as very primitive primates, tending to connect this order with unguiculate mammals; and secondly, because Bolk (1916) has found in the dentition of the Hapalidæ a supposed solution of the origin of certain phenomena of the human dentition.

It may be stated at the outset that in the writer's opinion both these conclusions are very far from the truth; that the Hapalidæ are

in reality less primitive than some of the Cedidæ, that they are widely remote from the origin of any of the Old World series, including man, and that Bolk's comparisons are largely based upon independently acquired features.

Repeated comparisons of the skulls of the Hapalidæ (*plates 12, 13*) with those of the Cebidæ have finally led to the conclusion that the former have been derived from the ancestors of *Aotus* ("*Nyctipithecus*") and *Callicebus*, of the Cebidæ, from which genera they differ chiefly in the enfeeblement of the lower jaw and zygomatic arch, the loss of the upper and lower third molars, the reduced size of the orbits, the varying anteroposterior elongation of the cranium and the enlargement of the canines. The molar series (*pl. 13*) both upper and lower, are more or less retrogressive in size and probably also in the form of the crown. Starting with *Aotus*, of the Cebidæ, and passing to *Tamarin* (*Seniocebus*) and the other genera of the Hapalidæ, we may observe the traces of this retrogressive process. In *Aotus* the first upper molar, which in most mammals is conservative, in form, retains the subquadrate contour which is characteristic of this tooth in other Cebidæ; but the second upper molar is reduced in size and subtriangular in form, through the partial suppression of the posterointernal corner. The third upper molar is very retrogressive, only the anterior moiety remaining, the posterior moiety being barely, if at all, represented. The Hapalidæ have carried this process much further, suppressing the third molars entirely and often reducing the second molars to a form quite like that of the third molars of *Aotus*. Even the first molars of the Hapalidæ apparently have shared in this general retrogressive evolution, for in *Midas* the metacones of the first molars are reduced in size, and the hypocones are vestigial, a condition which, in view of the generally high grade of organization of the skull of this animal and of the advanced development of the molars in many Eocene primates, we may not safely regard as primitive. The roundness of the molar cusps and of the whole crowns is also a retrogressive character in certain other phyla of primates, including the human phylum.

The Hapalidæ, having certain peculiar specializations, also parallel different members of the Cebidæ in different characters. In the extreme lengthening of the cranium, as seen from above, *Midas* (*pl. 13*)

of the Hapalidæ parallels and even surpasses *Ateles*, *Cacajao* and *Pithecia*, and approaches *Saimiri* (*Chrysothrix*) of the Cebidæ. In the sharp protrusion of the interorbital and nasal region they go beyond *Aotus* and *Callicebus*, as also in the retraction of the face beneath the cranium (*pl. 12*). As in these genera, the malar walls of the orbits are broadly convex and have much the same relations with the zygomatic arches. It is quite possible that the Hapalidæ may have been derived from nocturnal or seminocturnal ancestors with enlarged orbits, much like those of *Aotus*, and that their still protruding orbits have become secondarily reduced in size. The ramus of the lower jaw (*pl. 12*) is much more slender and the angular process more distinct, than in *Aotus* and secondarily suggests the conditions in *Chrysothrix*, which genus the Hapalidæ also approach in the enlargement of the canines and of the premolar series.

As noted above the Hapalidæ have but two upper molars, the third having been lost and even the second greatly reduced. The small size of the third molar is suggested in various genera of the Cebidæ, especially *Saimiri;* but as the other resemblances with the latter are not very close, the reduction of the third molar may be regarded as a parallel adaptation to the extreme lengthening of the braincase and the forward crowding of the whole dental arch. It is as if the center of maximum growth had shifted forward from the molar to the antemolar region. The three upper premolars (*pl. 13*) are wide and bicuspid, and the tritubercular upper molars have low rounded cusps, the dentition as a whole being adapted, as in so many other small primates, to a mixed diet of fruit and insects.

In brief, the skull and dentition of the Hapalidæ appear to indicate that its nearest relatives are *Callicebus* and *Aotus* of the Cebidæ and that it has paralleled the other Cebidæ in various ways.

The question of the derivation and relationships of the Hapalidæ has such a direct bearing upon the problem of the evolution of the human dentition that it seems necessary to consider briefly some pertinent morphological evidence other than that which is to be found in the characters of the skull and of the dentition. Gidley, following Wortman, is inclined to regard the Hapalidæ as a "rather primitive and generalized group," partly because their pollex is said to lack the usual opposability typical of the primate thumb, and he concludes

(1919, p. 276) that these "small, lighter-bodied animals seem never to have acquired the function of grasping a limb, but depend rather on their sharp, widely-spread claws for support in progressing among the treetops." If this conclusion were correct the Hapalidæ would be a structural link between the primates and more primitive clawed mammals. But a comparison of their hands and feet leads me to the opposite conclusion that their "claws" are merely bent up nails, such as are already foreshadowed in less extreme condition in the smaller Cebidæ (including *Callicebus* and *Aotus*), and that the lack of opposability is a degenerate and secondary character.

The first consideration is that true unguiculates that climb have short proximal phalanges and powerful flexors of the digits, by means of which they are able to dig their claws into the bark of trees; while the Hapalidæ have the longer and more slender metatarsals and phalanges, and the feeble flexors, of the primates, which depend rather on grasping the branches with the friction pads on their digits.

Secondly, the hallux of the Hapalidæ is characteristically primate in form, bearing the flattened nail which is found even in Eocene primates.

Thirdly, the fibula of the Hapalidæ shows a tendency to be immovably jointed or partly coalesced with the tibia at the lower end, a condition which no morphologist could safely regard as primitive.

Fourthly, the backbone and pelvis of the Hapalidæ are in many respects more or less intermediate in form between those of the primitive Eocene Notharctidæ, on the one hand, and of the larger Cebidæ on the other, a fact indicating the essentially primate character of their whole locomotor apparatus, and strengthening the conclusion that the clawed condition of their digits is entirely secondary.

This digression seems necessary in order to establish the true position of the Hapalidæ as aberrant members of the New World series. Their very small size is possibly secondary and they have suffered a serious impairment of the typical primate method of locomotion. But they are extremely progressive in the great enlargement of the braincase and in the shortening of the dental arch posteriorly, in the consequent loss of the third molars, and in the reduction of the second; also in the progressive development of the upper canines, which are large and widely separated and well able to pierce the bodies of still smaller or more helpless animals, such as young birds and insects.

Bolk apparently leaves out of consideration the wide differences in skull and skeleton between the Hapalidæ and any of the Old World series, including man. The Hapalidæ, like the Cebidæ, have:

(1) The bony auditory meatus (*pl. 13*) in the form of a large ring, which is short in its transverse diameter, widely open at the outer end and greatly expanded anteriorly on the inner end, so as to overlap the inflated bulla. Behind the ring, between it and the underlying bulla, is a conspicuous foramen for the internal carotid artery. In all the Old World series, on the contrary, the tympanic bone forms a narrow tube, projecting transversely and more or less overlapped between the postglenoid and the post-tympanic processes. Its inner end is not greatly extended anteriorly where it overlaps the bulla.

(2) The skull as seen from above in the Hapalidæ (*pl. 13*) and the rest of the New World series (*plate 10*) is very long and narrow, with a marked posterior prolongation. In the Old World series, on the contrary, the cranium is primitively short and wide, not bulging backward in the occipital region except in the Hominidæ, where a similar condition is likewise correlated with the secondary overlapping of the cerebellum by the cerebrum.

(3) The eyes in the Hapalidæ (*pl. 13*) and most other New World monkeys (*plate 10*) are directed outward and forward, whereas in the Old World series they are directed forward, and the crests above the orbits tend to be arranged at right angles to the long axis of the skull.

(4) In the New World series the top of the braincase typically lies considerably above the dorsal rim of the orbits (*plates 8, 12*), while in the Old World series this dorsal rim is usually nearer the level of the top of the cranium except in obviously specialized end members, such as the orang, and still more, man, where there has been a rapid increase in the vertical diameter of the braincase.

(5) In the Hapalidæ, as in the less specialized Cebidæ (*e.g., Callicebus*), the maxilla (*pl. 12*) beneath the orbit is shallow, while in all the Old World series, including man, it is much produced forward and downward below the level of the orbits.

(6) The nasal region of the Hapalidæ is of the platyrrhine type, with the nasals widely separated at the base and tending to face outward ("narines éloignées," De Blainville); whereas in the Old World,

or catarrhine, series the opposite nostrils tend to be drawn together toward the midline so as to form a narrow V ("narines rapprochées").

Further, the anatomy of the Hapalidæ as described, for example, by Weber (1904, p. 784), leaves no doubt that they belong in the platyrrhine series, and affords no evidence either for any annectant position between primates and unguiculates, or for the view that they represent a structural starting-point of the Old World series.

DO THE CEBIDÆ AND HAPALIDÆ AFFORD STRUCTURAL STAGES IN THE EVOLUTION OF THE HUMAN DENTITION?

The important evidence derived from taxonomic considerations wholly fails to support Bolk's view that the loss of the third molar in the Hapalidæ is peculiarly significant in the problem of the origin of the human dentition. Bolk (1916, p. 139) supposes that man, and presumably the whole Old World series, passed through a stage still represented in the Hapalidæ, in which the last molar, both in the upper and lower jaws, was normally suppressed and in which the last deciduous molar became retained as the first permanent molar of the human dentition. The occasional appearance of a fourth molar would therefore be explained as a reversion to a pre-hapalid stage and as a reappearance of the suppressed m^3 and m_3.

That the first permanent molar of typical placental mammals is serially homologous with their last deciduous molar has long been believed by Matthew and others, and is supported by the fact that in very numerous phyla of mammals the first permanent molar erupts early, is closely associated with the last deciduous molar, and resembles it so closely that a pure convergence in origin and pattern seems highly improbable. But we assuredly do not need to cite the Hapalidæ in this comparison, which holds true apparently in all phyla of placental mammals that retain a normal replacement of the dentition.[4] The conditions presented in the Eocene and modern lemurs, as well as in many other phyla of mammals, tend to indicate, in the opinion of the writer, that not only the first permanent molar but also the second and third are serially homologous with the deciduous molars, and

[4] Evidence for this statement is cited in my "Studies on the evolution of the Primates," 1916, pp. 243–246.

that the replacing teeth of the molar series were eliminated as far back as the cynodont reptilian stage;[5] first, because an examination of available material indicates that as far back as the cynodont stage there was but a single set of molars without successors; secondly, the nearness of the first molar with the last deciduous molar in time of eruption is not significant of any special homology which would not also apply to the remaining molars, for the reason that in both Eocene and modern lemurs the second and third molars appear shortly after the first molar, and the great delay in the appearance of these teeth in later forms is perhaps secondary, conditioned often by their bulk, which is too great to be accommodated in the infantile jaws.

According to Bolk's view the third molars of the catarrhine series are not homologous as a whole with those of the Platyrrhinæ; consequently the third lower molar in all the Tertiary Old World primates as far back as the primitive *Propliopithecus* and *Parapithecus* are not homologous with the third molars of existing Cebidæ. On palæontological grounds this must be considered a wholly unproved assumption, since there is nothing to indicate that the third lower molar of the ancient *Parapithecus* and *Propliopithecus*—Old World genera of extreme primitiveness—is not homologous with that of other primates, which it resembles in essentials.

How, then, are we to regard the fourth molar of man and other primates, and what is its origin? Is it, as Bolk supposes, a reversion to a pre-Hapalid stage, before the loss of the true third molar? A fourth molar, especially in the lower jaw, is not infrequently recorded in the orang and even in the gorilla and other anthropoids.[6] It is also recorded in *Otocyon*, among the Canidæ, and in the lemurs, and I have before me a lower jaw of the platyrrhine genus *Ateles* (Amer. Mus. no. 17,218), with a small but well developed fourth molar on each side, directly behind the third molar.

It is by no means necessary to regard these fourth molars as reversions to a pre-placental or marsupial condition. Granger, of the American Museum of Natural History, has collected, examined and catalogued thousands of specimens of American Paleocene and Eocene placental mammals, belonging to all the known orders and fami-

[5] See Part I.

[6] Even a fifth lower molar has been observed by Hellman (1918) in the orang.

lies, but he has never, he informs me, observed a single instance of the presence of a fourth molar in these very early placental mammals. Matthew testifies to the same effect, and so far as I am aware the extensive literature of the Eocene placental mammals of America and Europe affords no record of a fourth molar.

The presence of a fourth molar in modern mammals may be ascribed to a specialized or relatively new tendency of the dental lamina to bud off more than the normal number of teeth. This tendency is very obvious in certain phyla of mammals, such as the Sirenia and armadillos, where the number of molar teeth is considerably increased, and in certain odontocete Cetacea where the primitive number of cheek teeth is multiplied.

V. SUMMARY AND CONCLUSIONS FOR PART III

In spite of the fact that probably none of the animals studied in Part III lie in or near the direct line of human ascent, yet they show us successive improvements in the skull and in the dentition which mark a general advance from the lemuroid toward the anthropoid grade of evolution.

The lorises and galagos represent an early attempt to evolve a large-brained large-eyed primate out of the primitive lemuroid stock, but they still retain the lemuroid face and their cheek teeth do not advance much beyond the primitive lemuroid types with tritubercular upper molars. At the same time they parallel the true lemurs in the specialization of the incisors and canines for combing their fur, and thus they remove themselves from the line of human ascent.

The tarsioids as a group equal or excel the lorises and galagos in the enlargement of the eyes and braincase, and some of them foreshadow the Platyrrhinæ in the reduction of the dental formula to $I\frac{2}{2}\ C\frac{1}{1}\ P\frac{2}{2}\ M\frac{3}{3}$. They finally attain a high and almost pithecoid grade of organization in the construction of the orbits, auditory, and basicranial regions; their upper molar crowns tend to pass from a wide trigonal to a subquadrate form, divided into subequal anterior and posterior moieties as in the Old World stock; while their lower molars, at first with small trigonids and wide talonids, often lose the paraconid and tend to develop a hypoconulid (as in *Microchœrus*). But,

as a group, they carry too far the enlargement of the orbits and internal ears, and mostly acquire specialized conditions of the front teeth which rule them out of the line of ascent either to the Platyrrhinæ or to the Catarrhinæ.

The platyrrhine or New World series seem to have started from small primates which had a tarsioid form of skull, but with certain marked differences in the front teeth and in the region of the auditory bullæ, which are more compatible with derivation from primitive members of the Notharctidæ. These presumably primitive and "tarsioid" ancestors seem to be most nearly represented in the present fauna by the genus *Callicebus*, a small cebid monkey once classed with the marmosets, but much more primitive in many characters of the skull and dentition. From this primitive type we traced an "adaptive radiation" (illustrated in *plates 8–11*) along several lines:

The first line, represented by the large-eyed and nocturnal douroucolis, or owl-faced monkeys (*Aotus*, "*Nyctipithecus*") and possibly also by the marmosets (*plates 12, 13*) which appear to be more closely related to the douroucolis than to other Cebidæ;

The second line, represented by the relatively large and in certain respects highly specialized "howlers" (*Alouatta*);

A third line leading through or near the woolly spider monkeys (*Brachyteles*) and culminating in the true spider monkeys (*Ateles*);

A fourth and a fifth line represented by the aberrant uakaris (*Cacajao*) and sakis (*Pithecia*), which have procumbent front teeth but somewhat different types of grinding teeth;

The sixth and seventh lines, including the large-brained sapajous (*Cebus*) and the squirrel monkeys (*Saimiri* = "*Chrysothrix*"), which are by far the most advanced and pithecoid of all the Platyrrhinæ. The latter two genera have contributed to the wholly erroneous view of some authors (Ameghino, Sera, et al.) that the remote origin of the Old World series (including man) must be sought in the New World Platyrrhinæ. Nevertheless they afford an instructive example of the manner in which a pithecoid type of skull and dentition may have been derived from a more primitive tarsioid stage.

The marmosets (Hapalidæ), it was shown, are dwarfed Platyrrhinæ, derived perhaps from the ancestors of the douroucolis (*Aotus*), which have probably suffered retrogressive modifications of their locomotor apparatus and have become pseudo-unguiculates, climbing by means

of their claw-like, bent-up nails, and tending to lose the typical platyrrhine grasping adaptations of the hands and feet. At the same time they have lost the posterior or third upper and lower molars, a peculiar specialization which, together with many others, wholly unfits them to be structurally ancestral to the Old World primates. And yet Bolk bases an elaborate theory of the origin of the human dentition partly upon the conditions observed in the Hapalidæ!

The Platyrrhinæ as a whole afford a beautiful illustration of a natural group, the members of which show markedly diverse specializations, which have, however, not gone far enough to obliterate an underlying heritage received from the stem forms of the whole group. Unless one chooses to believe in the special creation of each genus of Cebidæ and Hapalidæ, the evidence is decisive that the "heritage" which they all have in common proves their descent from a common ancestral stock. This conclusion is evidently valid in spite of the almost complete absence of palæontological proof. Thus, when we have satisfied ourselves that we are dealing with a natural group, we thereby postulate the existence of a common ancestral species or genus, bearing distinctly primitive characters which have been lost by its diverse descendants, and other primitive characters which have been preserved by them. Now, the determination of these characters requires close and repeated examination of all the existing genera of the group. Thus we again perceive the indispensability of thorough taxonomic study in close connection with all investigations of the evolution of particular structures, such as the dentition.

Two of the most easily recognizable items of the platyrrhine heritage are the presence of three more or less bicuspid premolars on each side both in the upper and in the lower jaws, and the peculiar relations of the tympanic ring to the auditory bullæ (see below, p. 421). These characters, along with many others, separate all the Platyrrhinæ from the whole Old World series and should have been taken more fully into account by those who have sought to find the remote ancestors of man, and the ancestral conditions of the human dentition, among large-brained Platyrrhinæ.

(End of Part III)[7]

[7] The manuscript of the concluding part of this review has been received for publication in the December issue of this JOURNAL. The bibliography for all the parts will be appended to the concluding section.—(Ed.)

PLATES 8–13

(GREGORY: EVOLUTION OF THE HUMAN DENTITION)

FIG. 153. *Aotus*, × 1

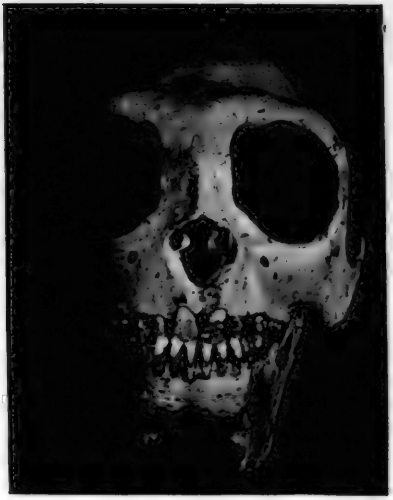

FIG. 154. *Callicebus*, × 1

FIG. 155. *Alouatta*, × 3/4

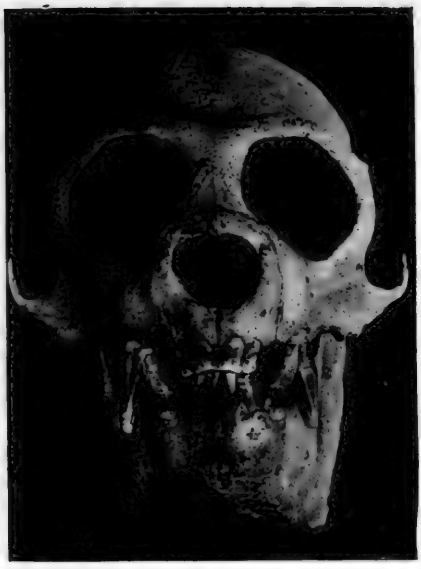

FIG. 159. *Pithecia*, × 1

FIG. 160. *Cacajao*, ×

FIG. 156. *Brachyteles*, × 4/5

FIG. 157. *Ateles*, × 4/5

FIGS. 153 TO 162, INCLUSIVE. COMPARATIVE SERIES: SKULLS

Skulls after Elliot, rearranged. Black lines indicate relat

FIG. 161. *Cebus*, × 4/5

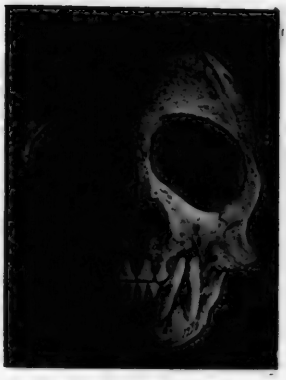

FIG. 162. *Saimiri*, × 1

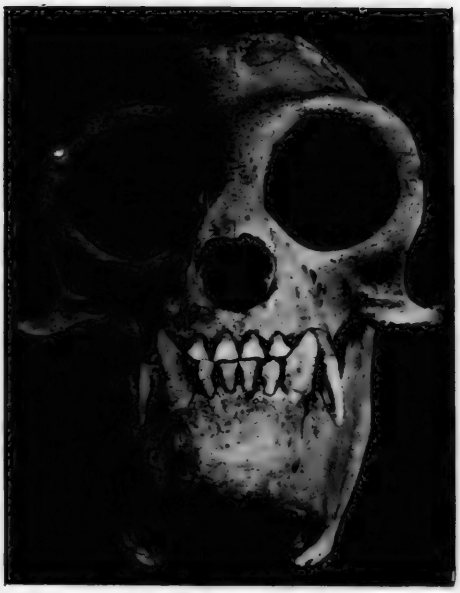

FIG. 158. *Lagothrix*, × 4/5

OF CEBIDÆ

onships.

FIG. 163. *Aotus*, × 1

FIG. 164. *Callicebus*, × 1

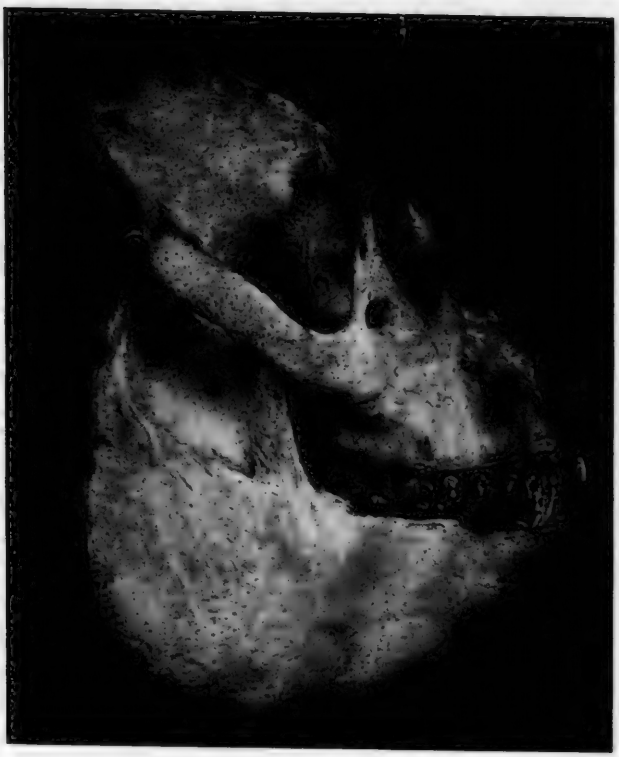

FIG. 165. *Alouatta*, × 3/4

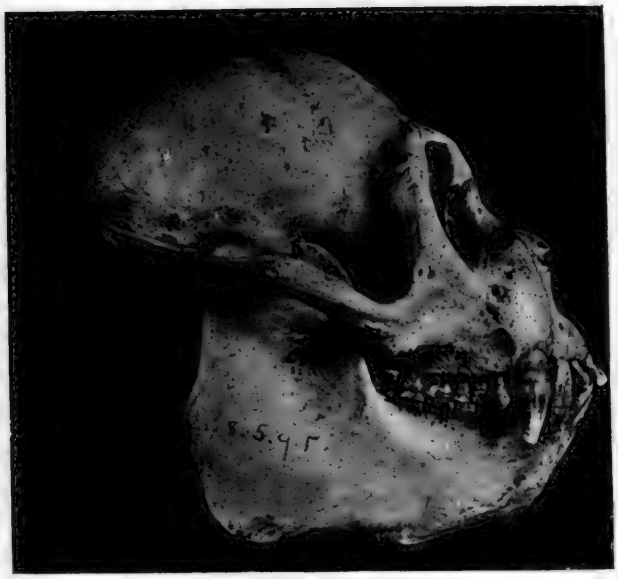

FIG. 169. *Pithecia*, × 1

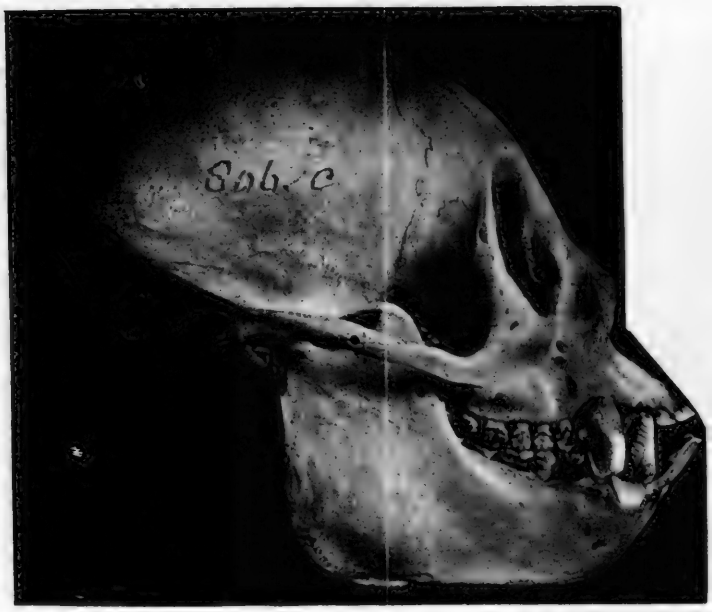

FIG. 170. *Cacajao*, × 1

FIG. 166. *Brachyteles*, × 4/5

FIG. 167. *Ateles*, × 4/

FIGS. 163 TO 172, INCLUSIVE. COMPARATIVE SERIES: SKULLS OF CEBIDÆ—*(Continued)*
Skulls after Elliot, rearranged

FIG. 171. *Cebus*, × 1

FIG. 172. *Saimiri*, × 1

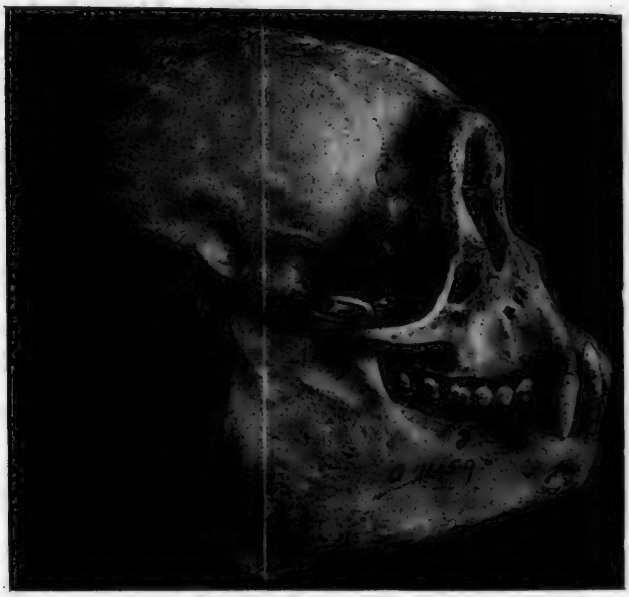

FIG. 168. *Lagothrix*, × 4 / 5

FIG. 173. *Aotus*, × 1

FIG. 174. *Callicebus*, × 1

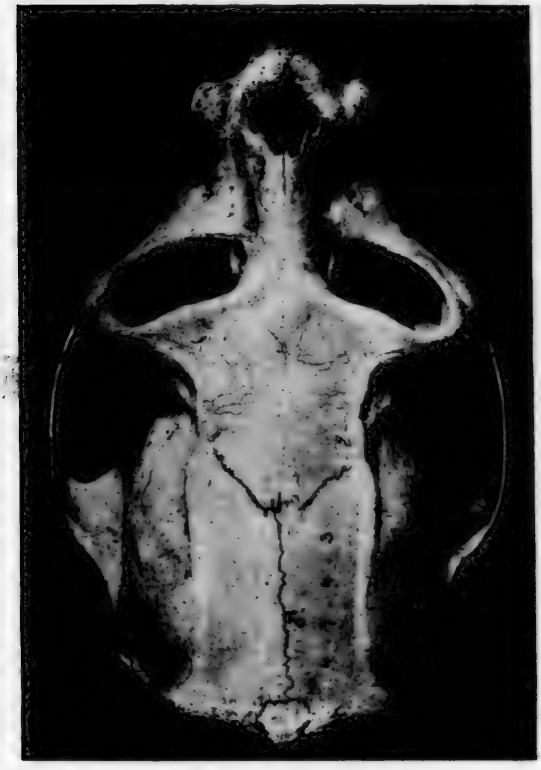

FIG. 175. *Alouatta*, × 3/4

FIG. 179. *Pithecia*, × 1

FIG. 180. *Cacajao*, × 1

FIG. 176. *Brachyteles*, × 4/5

FIG. 177. *Atel*

FIGS. 173 TO 182, INCLUSIVE. COMPARATIVE SERIES: SKULLS OF CEBIDÆ—(*Continued*)

FIG. 181. *Cebus*, × 4 / 5

FIG. 182. *Saimiri*, × 1

× 4 / 5

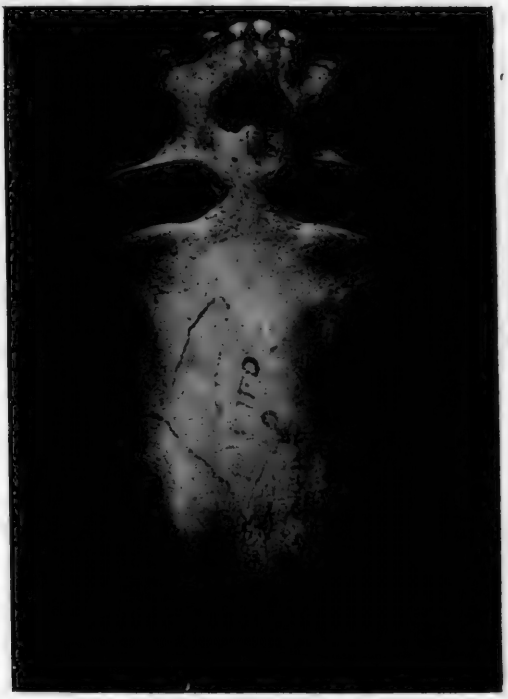

FIG. 178. *Lagothrix*, × 4 / 5

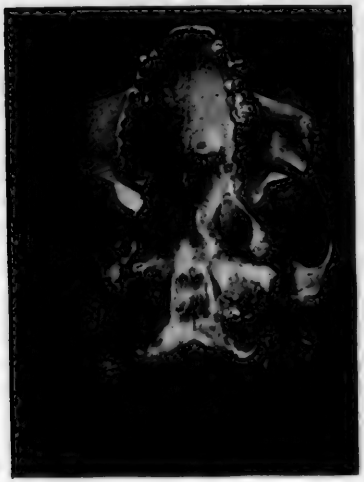

FIG. 183. *Aotus*, × 1

FIG. 184. *Callicebus*, × 1

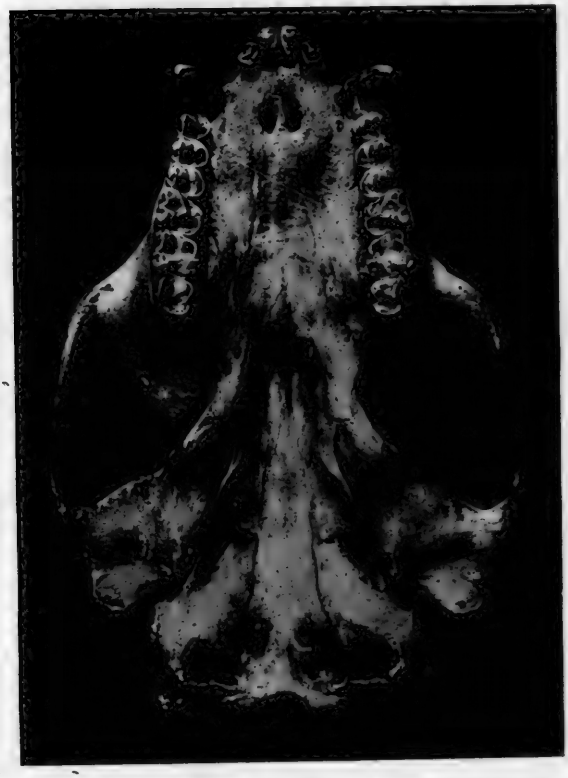

FIG. 185. *Alouatta*, × 3/4

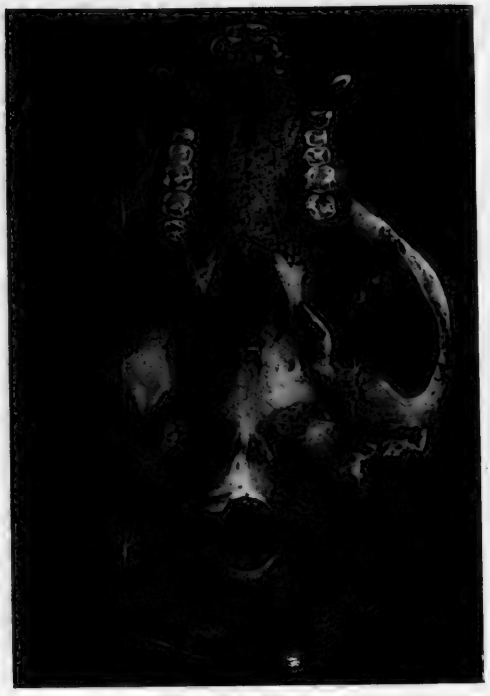

FIG. 189. *Pithecia*, × 1

FIG. 190. *Cacajao*, × 1

FIG. 186. *Brachyteles*, × 4/5

FIG.

FIGS. 183 TO 192, INCLUSIVE. COMPARATIVE SERIES: SKULLS OF CEBIDÆ—(*Contin*

Skulls after Elliot, rearranged

FIG. 191. *Cebus*. × 4/5

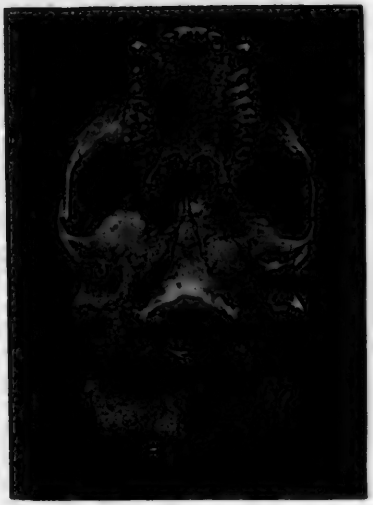

FIG. 192. *Saimiri*, × 1

7. *Ateles*, × 4/5

FIG. 188. *Lagothrix*, × 4/5

ed)

FIG. 194. *Callicebus*, × 1

FIG. 193. *Aotus*, × 1

FIG. 197. *Seniocebus*, × 3/2

FIG. 196. *Midas*, × 3/2

FIG. 195. *Oedipomidas*, × 3/2

FIGS. 193 TO 202, INCLUSIV

FIG. 199. *Callicebus*, × 1

FIG. 198. *Aotus*, × 1

FIG 202. *Seniocebus*, × 3/2

FIG. 201. *Midas*, × 3/2

FIG. 200. *Oedipomidas*, × 3/2

PLATE 12

FIG. 203. *Aotus*, × 1

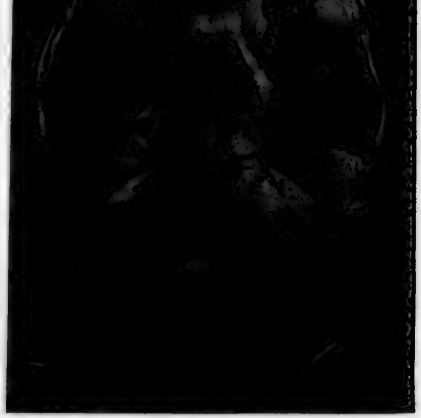

FIG. 204. *Callicebus*, × 1

FIG. 205. *Oedipomidas*, × 3/2

FIG. 206. *Midas*, × 3/2

FIG. 207. *Seniocebus*, × 3/2

FIGS. 203 TO 212, INCLUSIVE. COMPAR

Sku

FIGS. 203 TO 212, INCLUSIVE. COMPARATIVE SERIES: SKULLS OF HAPALIDÆ AND PRIMITIVE CEBIDÆ

Skulls after Elliot, rearranged

FIG. 205. *Oedipomidas*, × 3/2

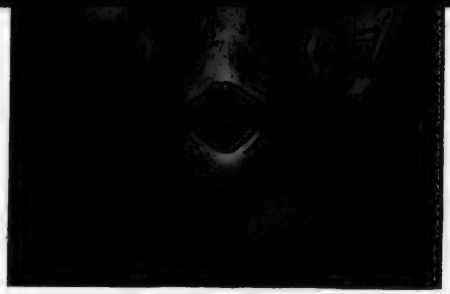

FIG. 206. *Midas*, × 3/2

FIG. 207. *Seniocebus*, × 3/2

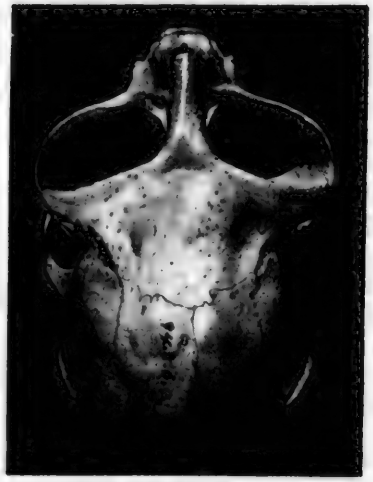

FIG. 203. *Aotus*, × 1

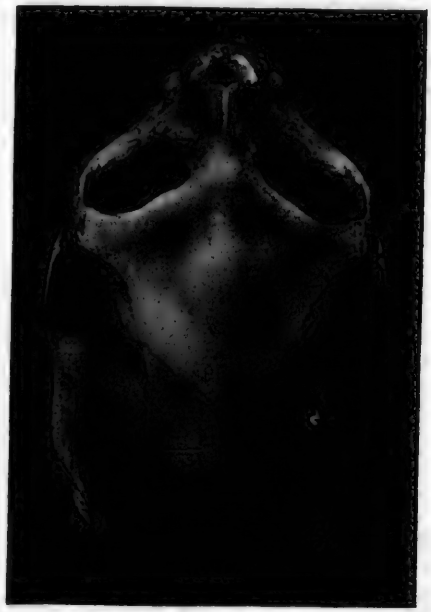

FIG. 209. *Callicebus*, × 1

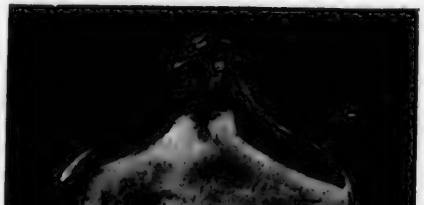

FIG. 210. *Oedipomidas*, × 3/2

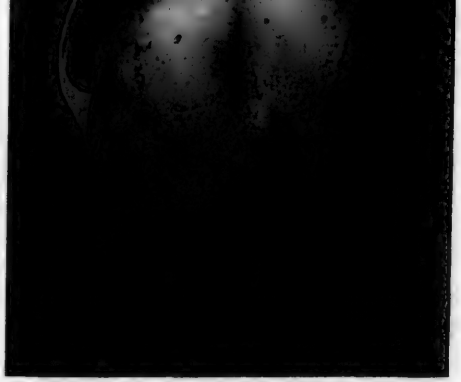

FIG. 211. *Midas*, × 3/2

FIG. 212. *Seniocebus*, × 3/2

A PLEA FOR A STANDARDIZED TECHNIQUE FOR ORAL RADIOGRAPHY, WITH AN ILLUSTRATED CLASSIFICATION OF FINDINGS AND THEIR VERIFIED INTERPRETATIONS[1]

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I. INTRODUCTION

Oral radiography has been regarded as a more or less tentative science. In fact, it may be questioned whether it has been looked upon as a science at all. By many it has been considered a sort of "extra motion," which may be taken to improve, but not very materially, the work of the dental practitioner. Its great possibilities have been sensed by some dental practitioners, however. That these possibilities are attainable, even remotely, has been conceded by only a few of the more enthusiastic. It is significant, however, that these enthusiasts are the practitioners who use radiography most generally and who are in closest touch with the rapid advances the science has made in the past few years.

[1] Read before the Missouri State Dental Association at its fifty-fifth annual meeting in Kansas City, Mo., on April 12, 1920.

[2] The names of the author's collaborators are given on pages 470 and 484.

In harmony with the findings of my dental collaborators, I maintain that oral radiography has reached a stage of development where, by a standardization of its technique, it can and does produce radiograms that can be relied upon as the physical basis for approximately 75 per cent of the correct diagnoses of all dental lesions. Not only does such standardization bring out clearly and unmistakably the facts about conditions that may be suspected by the clinician, but it also brings to light, in numerous cases, conditions that are wholly unsuspected. It gives the clinician what should be the very starting point of his diagnosis. My dental collaborators assure me that no dental diagnosis ever should be concluded until accurately made radiograms of the case have been obtained and studied. In short, I believe the best informed dentists now agree that accurately made and properly interpreted radiograms are among the fundamentals of scientific dental practice.

Since standardization of technique is the cornerstone of scientific radiography, it shall be my endeavor, in this paper, (a) to make clear just what is meant by "standardized technique;" (b) to present some of the important means whereby it can be attained; and (c) to tell something of the supplementary research we have conducted to test the reliability of the interpretations of our radiographic findings.

Radiographic technique, to afford a reliable basis for diagnosis, should include (a) a series of exposures made without photographic variations, in order to insure uniformity of results for accurate comparison of the normal parts of the mouth with the diseased parts; (b) a procedure so accurate that radiograms of the given parts can be made at any later period, without photographic or technical variations from the first series; (c) the establishment of a radiographic normal exposure, as explained below; (d) the means (after the first series of exposures, if there appear to be pathological or physiological changes that the established exposure has not shown in sufficient detail) of making additional exposures by varying the time factor only; and (e) a procedure whereby every finished set of radiograms shows the teeth in their true position and anatomical relation, to each other and to the adjacent structures of the face.

Unless such a uniformly dependable technique is used, it is practically impossible to follow a case radiographically with any semblance

of scientific accuracy. It stands to reason that radiograms of such technical uniformity can be made only by those who specialize in this particular branch of science and make it their life work. Radiography, as a diagnostic aid, is valuable in proportion to the amount of experience the radiographer has gained, both from association with clinicians and from research to determine the structural variations and changes in tissue that are shown by radiograms. The highest degree of technical expertness, it is needless to say, is required in order that the radiographer may render the most perfect service, both in making the radiogram and in interpreting it.

In the early period of my work, I tried to make uniform exposures and reproduce them at later intervals, but with little success, except in a few cases. Since that time, I have used several types of equipment, but these were discarded as unsatisfactory for the purposes of a standardized technique. Four years ago, I took up this question of equipment with the engineers of a firm that builds roentgen-ray machines. Together, we worked out a plan that called for the installation of equipment which would deliver, at all times, a uniform stabilized flow of current, with dark-room and operating rooms so situated, ventilated, and systematically arranged, that there was a possibility of approaching standardization. After the installation was completed, there remained many more factors to be worked out before we could even hope to attain a standardized system. During this experimental period, I made all the exposures, mixed all the chemicals used, supervised the cutting of all the films, did the developing of all the negatives, and carried on many experiments in an attempt to devise a system that would have a minimum number of variable factors.

It is my belief that this technique, as now evolved, can properly be called standardized, for, with it, we have *reproduced* sets of radiograms after intervals of three years or more—the second sets have virtually the same appearance as the radiograms of the first sets. Furthermore, I have tried out this technique in other laboratories having similar equipments, and the results obtained, after some experimental work was done, were uniform and without photographic variation.

We have developed scientific interpretation of radiograms through extended supplementary research and clinical verifications. Material from many cases, showing variations from the normal, has been studied bacteriologically, such as apices of devitalized teeth, teeth having exposed pulp, various types of tissue taken from areas where teeth had been extracted, cysts, and diseased and normal bone. Soft and osseous tissues have been examined surgically, and the structural conditions and changes noted and compared with the corresponding radiographic findings.

In 1914, Dr. Josef Novitzky (4, 5, 6) insisted that the radiographic work then being done was not showing many of the pathological conditions which he thought could be shown by improvements in technique. This encouraged me to try for more accurate results. He gave me the benefit of his impartial opinion concerning the clinical and surgical findings of cases upon which he operated and for which I had made the radiograms. This hearty coöperation helped me to correct many errors in procedure.

Later, a two-year period of preliminary research work, with the assistance of Dr. Joseph A. Pollia, proved to our satisfaction that the *accurately* made radiograms did not lie. Our improved radiography began to show, more clearly, changes in bone that were not theretofore recognizable by us. A new period of research was then begun, this time with authoritative investigators and improved technique. The work has been done under the general supervision of Dr. Karl F. Meyer of the George William Hooper Foundation for Medical Research. Mr. C. Coleman Berwick, A.M., is the collaborating bacteriologist. This work is still in progress. Reports of the findings will be published. One result of this work has been already announced—the utility of crystal violet and brilliant green as an agent for disinfecting the oral mucosa before operating surgically and as a bacteriostatic for post-operative treatment (1).

The results obtained in these various researches have been turned back into our laboratory, and have enabled us to effect improvements in our radiographic technique and interpretations. I present below the major features of our procedure.

II. A STANDARDIZED TECHNIQUE

A standardized technique is the net result of a coördination of many factors. The various steps must be adjusted perfectly. The failure of any one of the factors means failure to attain standardization.

The basic conditions on which the radiographer proceeds must be as nearly perfect as possible. His whole establishment must be planned to eliminate confusion, to save time, and to make for a systematic handling of patients. The psychological effect upon the patient must be considered. The furnishings should be restful, and arranged in good taste. This helps to inspire the patient with trust and confidence. It is important that the patient be impressed with the assurance that he will receive the best service possible; and nothing helps in this respect so much as the first impression given by a comfortable, well appointed, suite of offices, well aired and cleanly.

The patient should have been told by the dentist, before being referred to the radiographer, that there is some discomfort and possibly some pain connected with the process of making the radiograms. Furthermore, the dentist should have explained to the patient the great importance of good radiograms in reaching a correct diagnosis; and the importance of this diagnosis to his (the patient's) future health. Good radiograms, it should also have been explained, can be obtained only with the direct coöperation of the patient.

I find that few dentists give their patients these hints before referring them to radiographers. Dentists are apt to tell their patients, in off-hand fashion, that "it takes only a minute and doesn't hurt" to obtain a radiogram. The facts should be explained to the patient, in a confident and careful way, in order to win, first, the interest of the patient and, later, his willing coöperation.

Fear is the greatest initial difficulty to be overcome. If one holds the patient's confidence long enough to get the first exposure, however, the rest can be obtained. Nervous patients are usually intelligent and, while they are hard at first to handle, they know how to coöperate because they comprehend quickly. Children are not difficult. They have the curiosity of childhood and wish to know the reasons for everything. It saves time to explain to them just

what is going on. Then firmly insist that they help by holding perfectly still. They usually do. There is demand here for speed, absolute assurance in the physical handling of the patient, and, so far as possible, noiseless manipulation of the tube and machine.

I have found that the inverted lighting system is best, for all the rooms of the offices and laboratory. It is softer, less irritating to the eyes and nerves of both patient and operator, and helps, to a great degree, to relieve the minds of patients of reports they have heard about the harmful effects of the roentgen ray. It is most important to have the noise-producing parts of the equipment behind closed doors outside of the laboratory operating rooms. There is then no disturbance of the laboratory calm, and no sparking is heard. Even healthy adults, not unacquainted with the x-ray process, appreciate this refinement.

Evidence of the careful observance of sanitary methods should be apparent. Easily washed enamel fittings should be used. Waxed paper should be used upon the handles on the control table, these to be changed for each patient. It is advisable to have a large wash basin, controlled by easy-working levers touched by a foot, at which the patient washes his hands carefully before holding the films in his mouth. The operator, of course, should do likewise.

All this, necessarily, is but a beginning. It is principally a recognition of, and concession to, the psychological factors in the so-called "art of handling patients."

The details of equipment are important. Three and a half years ago a 10-kilowatt Snook interrupterless transformer was installed. This was placed in a small room between two operating rooms. It has double-throw switches for both the overhead wires and for the wires connecting the transformer with the control tables in either operating room. These connections are run through the walls for the overhead wiring system. The wires to the control tables are run downward through the floor of the transformer room, then under the floor for about ten feet, and up through each operating-room floor to the control tables. Both the Snook transformer and the Coolidge-tube transformer are directly connected with the main current supply lines in the street by extra large wires which run through a private conduit. Double naught (00) wire is used for the Snook

transformer, and number fourteen (14) wire for the Coolidge-tube transformer. This is expensive, but it is necessary if one wants to avoid the many drops in line voltage that would occur if the transformers were connected with the service lines in the building, which are used by many other tenants. This heavy wiring eliminates all drops in line voltage, because it is of sufficient capacity to carry more current than will probably be used, and is insurance against loss of current by resistance. Stability and evenness of flow of current are basic factors and, with this installation, many exposures may be made as rapidly as one cares to make them, without over-loading of wires or transformers. The only uncontrolled factor is the change in line voltage that may develop at the main power-plant or sub-station. On Saturday afternoons, when many large users of current shut down their plants, this change is very noticeable and my meters fluctuate so much that it is almost impossible, then, to operate with accuracy.

To eliminate the open spark-gap, which is very noisy and disagreeable to have in an operating room, we installed the spark-gap meters for use with both gas and Coolidge tubes. This device has proved very accurate; but it is tested frequently by checking it with the open spark-gap on the transformer. In the control table of the Coolidge-tube room, an auto-transformer and control is installed to further eliminate fluctuations of current flow. If any fluctuation does occur, it may be instantly detected by either the spark-gap meter, volt meter, or milliamperemeter, all of which are in circuit. This fluctuation may be corrected or equalized by slight adjustment of the Coolidge-tube control, which is mounted beside the switch board and on which the operator holds his left hand, the right hand being used to manipulate the operating switch.

The 7-inch, fine-focus, Coolidge tube is used for intra-oral films, because it will stand longer exposures at shorter intervals with much less heating; consequently, it is more uniform in penetration than the small, radiator-type, Coolidge tube. I have used one 7-inch, fine-focus, Coolidge tube for over seventy thousand exposures, all of which have been for from two and a half to ten seconds; the tube is only slightly discolored, and the target is not pitted. I attribute this to the fact that the tube has never been allowed to become overheated, for 20 to 25 milliamperes is the maximum amount of current that is allowed to pass through the tube.

Gas tubes are used for making the extra-oral mandible plates and for all plates of the accessory sinuses. It is not possible to get as accurate a technique with the gas tubes as it is with the 7-inch Coolidge tubes, but a fair degree of standardization can be had with gas tubes if one will try. The first step is to select a tube that has been pumped, or whose vacuum has been exhausted to about the degree that will show a back-up spark of about the length that is required for the work to be undertaken. Great care must be used in seasoning this new tube. If a $4\frac{1}{2}$ inch back-up is required, season this tube for that back-up, never permitting more current to pass through the tube than is necessary to give this back-up. The gradual heating of the tube while seasoning and before using and the limiting of the amount of current in-put, will, after a time, season the tube to a point where one can say that it has almost a permanent back-up and penetration. If the tube raises in vacuum, use the reducer that is built on the tube, but do this carefully and take precautions against lowering the vacuum too fast, or the tube will become "freaky" and unreliable.

To standardize a Coolidge-tube technique, five factors must be kept constant: (1) Equipment of great enough capacity to deliver a uniform and stabilized flow of current to the tube; (2) definite settings for both the auto-transformer and rheostat dials that will give a 3-inch back-up spark and 20 milliamperes (found to be sufficient penetration for *all* patients); (3) a definite period of exposure, sufficient for the *majority* of patients, when exposures are made of the same corresponding area; (4) the distance of the anode of the tube from the film in the mouth, when exposures are made of the same area in each patient; (5) the speed of the emulsion on the film (in other words, the same *quality* of film must be used constantly). When these five factors have become regular procedure and are used in all cases, a normal exposure for one area has been established.

If the patient presenting himself for radiographic work is of an unusual type, in regard to the apparent heaviness of the tissues, one normal exposure may be made to determine the patient's deviation from the normal, or the experience of the radiographer may be ample to justify his making all the necessary exposures. In either case the only variation from the normal exposure will be the increased length of time of exposure. All the other factors may remain the same as with the average patient.

Having an exposure technique that is a definite procedure in all cases, and knowing that the equipment is adequate and stabilized and that the basic features are in coördination, how should the radiographer proceed to handle the average patient for an entire-denture case?

First, the extra-oral mandible plates are made. The patient lies upon a table, and is instructed to close and hold the jaws firmly together in normal occlusion; the mandible is placed as closely in contact with a glass plate as the thickness of the intervening tissues will permit. The plate rests on a flat metal tray, which is supported by pillows, thus making a plate holder that is adjustable. The various adjustments are maintained by using sand bags to support both the patient's head and the pillows.

There are so many types of mandibles that no set rule can be laid down, but the important thing to bear in mind in the making of extra-oral plates is to have the patient in such a position that the shadow cast by the hyoid bone is not superimposed over the mandible. With the average patient this may be accomplished by having the patient lie upon the shoulder and by adjusting the pillows supporting the plate, which is forced upward against the mandible. Then palpate for the hyoid bone and get its position, by putting the upper shoulder backward, bending the neck until the mandible lies parallel with the plate. The line of focus is thus from a point above and slightly to the rear and below the mandible, opposite to the side to be radiographed. By slight adjustments of the tube-stand, the rays may be projected above the hyoid without distortion of the lengths of the teeth or loss of the occlusal planes. Both sides of the mandible are radiographed for comparison.

For these exposures, 25 milliamperes, with a $4\frac{1}{2}$-inch back-up spark, is an ample amount of current. There are cases in which a softer tube may be used to advantage. In such instances we use a tube that will permit of a 4-inch back-up and 50 milliamperes.

The distance used for these exposures is 24 inches from the anode of the tube to the plate. The length of time must necessarily vary for this class of exposure because there are great differences in the thickness of the tissues; but six to eight seconds will produce clear negatives of the average patient. These plates are immediately sent to the dark room to be developed.

For the intra-oral films we use an Allison chair, which allows the patient to lie immobile in a horizontal position. The patient should be told to take several deep breaths to induce relaxation before placing the films in the mouth. With the film positioned as it should be, the patient should be instructed to put his or her finger tips upon it to hold it firmly in place against the teeth to be radiographed, and then to close the lower jaw firmly upon the fingers; not hard enough, of course, to cause soreness. This biting adjustment has three advantages: it prevents gagging, holds the film firmly in place, and gives the patient something on which to concentrate his attention. One must work hard for this coöperation. It represents half of one's success in getting the best possible results.

When working out a full-denture case, we make it a practice to expose all of the upper teeth first, starting from the incisors and working around to the molars, first in one direction, then in the other, using the number of films and positions as shown in the accompanying illustrations of entire-denture cases. (See *plate* 5, *figs.* 29 to 47.)

The films should be easily within reach, in all sizes, and in sufficient number. Here is a most important factor for the intra-oral exposures! We use six major sizes of film, for the various areas of the mouth, with three or four "freak" sizes to fit special formations occasionally encountered. Having these various sizes of film, specially adapted to particular areas, we are able not only to place them accurately for angle, but also to repeat such placings accurately, as they fit naturally into place.

While going through the primary stages of oral radiography, some seven or eight years ago, one of the first and most important difficulties was overcome by the realization that all of the films on the market were of one size, that they could not be used without being bent to fit the mouth, and that a bent film is photographically a distortion of the area exposed. We then began to cut and prepare our own films, experimenting with different sizes and shapes to fit the various areas of the mouth, using a German-silver plate on the back of each film to prevent bending. The results so far exceeded our expectations that we have never used the stock film since. We now have a large dark-room, built and equipped especially for the preparation of oral radiographic films.

The two upper corners of the film are rounded for the comfort of the patient; the two lower corners are left square, and extend out of the mouth far enough so that, in developing and mounting, the exposed portion of the film need not be handled or mutilated. In wrapping, a metal plate, the same size and shape of the film, is placed on the back of the film; the two are wrapped together in heavy, light-proof paper, and then wrapped again with white waxed paper as a sanitary measure and to exclude moisture. After each day's work the metal plates are sterilized and dried, and can be used again.

The metal plate backing the negative has other interesting and important properties: it stops the roentgen-rays from further penetration and acts as an intensifier on the negative. We have experimented with and without this metal backing, and have found that the negatives lose at least 20 per cent of their brilliancy when the metal backing is not used.

For any given area, a normal intra-oral exposure can be established. For illustration, the upper central incisor and lateral: a 3-inch backup spark, 20 milliamperes of current, distance of 24 inches, and timed at $2\frac{1}{2}$ seconds, may be regarded as the normal exposure. If the patient is young and probably not heavy-tissued, it would seem natural to shorten the time of exposure. This is not necessary, as this variation will be taken care of in the dark-room. If the patient is heavy-tissued, and the bone probably very heavy and more dense than the average, one normal exposure can be made and developed to determine this patient's variation from the normal; or the dark-room assistant may be competent enough to determine this variation from the appearance of the developing extra-oral plate. If the patient proves to vary greatly from the normal, the only factor that it is necessary to change is the time of exposure; otherwise all exposures should be made on the normal basis.

Timing exposures, until recently, has been a personal equation, because the devices manufactured for this purpose were cumbersome and not very efficient; but there are now on the market devices which, I believe, will be efficient. Counting has been the most accurate method for timing rapid work and I have found that, by frequently checking myself and also having visitors check my counting, one may become very accurate in timing exposures.

With my technique there are six major variations in the timing of the normal exposures; first, for the upper central and lateral, 2½ seconds; second, the upper cuspid, 3 seconds; third, the upper bicuspids, 5 seconds; fourth, the upper molars, 6 seconds; fifth, the lower incisors, 4 seconds; sixth, all of the remainder of the lower teeth, 5 seconds. These variations in timing are the result of experimental work as to tissue thickness and to the different distances that the tube is placed from the film, the upper molars requiring more time than the upper central incisors because the distance between the anode of the tube and the film is almost twice as great for the upper molars. It may seem to many that these exposures are prolonged and that the danger of the patient's moving during exposure is increased. This is true, but there are many advantages that out-weigh these objections.

With high-powered equipment, the tubes may be placed as far as four or five feet from the negative; and, with only a slightly longer exposure, clean-cut negatives may be made without blur or fog. The distances I have found most practical are 24 inches for the upper anteriors, 30 inches for the upper bicuspids, and for all the lowers; and from 36 to 40 inches for the upper molars. Although it takes more time to become proficient in sighting at these distances, it is easy with practice.

There are three other reasons for recommending long-distance exposures. First, many more exposures may be made, with less danger of burning the patient; because it is an established fact that the soft, short rays are the dangerous ones, and these are lost in the air before striking the flesh surface when long-distance sighting is practised. Second, this distance apparently acts as a filter for the short rays that have very little penetration. Only the deeply penetrating rays, with the greatest actinic action on the film, will travel this far and still have ample penetration value. The distance-technique, when used on long-bone work, gives beautiful negatives of the bone and also holds the fine details of the soft tissue. Third, this method prevents the occurrence of a shadow of the malar process over the apices of the upper molars, when exposures of that region are made.

The latter point has been the subject of some controversy and is worthy of elaboration. Let me propose this test. Have some one place a finger of one hand directly before you at a distance of, say,

six inches from one of your eyes; then a finger of the other hand about three-quarters of an inch directly behind the first finger. Using only one eye, to correspond with the focal point of the tube, try to see clearly, with that eye, the rear finger of the two. It is impossible. Now have the two fingers, remaining in the same relation to each other, drawn gradually farther away from the same eye. At a distance of about three and one half feet, you will be able to see the rear finger quite clearly after a very slight side motion of the hands. This corresponds perfectly with the position of the upper molar roots with relation to the malar process. The rays, in fact, are shot just under the malar process and strike the molar apices directly.

With the long distance technique, I have found that 90 per cent of the upper molar apices will be shown with practically no shadow from the malar process. In 70 per cent of the cases, all of the antrum floor, and the molar and bicuspid roots, will be shown in their true relative anatomical positions. The 10 per cent of partial failures are the cases in which the malar process is too prominent or the vault too low. In such cases, other expedients may be resorted to, such as the use of extra-oral plates made with special attention to the upper teeth; or intra-oral films, focussing the rays so as to project them through the orbit; or, by greater penetration, to project rays through the malar process itself.

With this method of making exposures, the system must be continued in the dark-room, where it will be found that there are other constant factors to be established in the process of developing the plates and films. The dark-room should be large, well ventilated, and so situated as to be accessible to the operating rooms. If possible, it should be entered from a light-proof passage-way, so that one may enter or leave the dark-room during development. The walls should be covered with lead for protection against penetration of rays from the operating rooms.

The tray system of developing and fixing is used, so that each plate and film may be studied as it develops. The trays of developer and fixing-bath are separated by a large deep tank of running water. The hands should always be rinsed after taking them out of either solution, as a small amount of "hypo" will ruin the developer. This tank is also used for washing all negatives. Cleanliness in the dark-room and of all utensils is essential.

A system must be devised for maintaining a standard temperature of about 62° F., for the developer. A thermometer can be left in the developing tray at all times, so that any change of temperature can be quickly noted and corrected. Our local climatic conditions enable us to maintain an even temperature in our dark-room as well.

All dark-room lights should be so regulated that there can be no change in the amount of light used. If a 25-watt light is used today and, after burning out, is replaced by a 50-watt light tomorrow, the uniformity of development will immediately be unbalanced.

The dark-room technician should be thoroughly trained in the entire system of standardization and, also, well coached in the interpretation of the finished radiograms. This will enable her to recognize, and to develop to show, many conditions to advantage which can be seen in the first stages of development. If there are any variations in the different exposures of one case, she can readily control them and thus add greatly to the attainment of uniformity. She should not be interrupted nor obliged to leave the dark-room during development, as it is very necessary for her to give it her entire attention.

Care should be taken by the dark-room technician to protect herself from any infection which might be carried by the wrappings around the film. After unwrapping, the hands should be thoroughly washed and dried before handling the films.

Our experiments with many radiographic developing formulae has convinced me that any standard Metol-hydroquinone developer which is accurately mixed, frequently renewed, well filtered and used at the proper temperature, will give satisfactory results.

We strive for uniformity of all negatives in a given case. This enables one to compare diseased with healthy bone. In many cases there will be a great difference between the two sides of the mouth. To get uniform results, the films for each corresponding area on opposite sides of the mouth must be given the same exposure, and developed in the same developer, at the same temperature and for the same length of time. For example, if two exposures of the upper right and left cuspids are the same, they will develop in the same length of time and will appear uniform.

By the systematic placing of films in the developing tray, it becomes a routine matter to look at those that went in first, next those that fol-

lowed; and to proceed in this order of observation as they develop. Thus, no one film is carried beyond the stage at which it should be taken out of the developer.

During development, the negatives should always be examined at a definitely determined distance from the red light. We have a hand-rest on our red light, on which to place our knuckles when the negatives are examined. If the negative is held four inches from the light at one time and one inch away at another, uniformity of development cannot be hoped for.

As stated above, the two extra-oral mandibular plates are exposed first and while these are developing, the twelve intra-oral films of the upper teeth are being exposed; and will be developed while the seven exposures of the lower teeth are being made. Thus, the developing is divided into three stages, allowing the dark-room technician ample time to exercise the necessary care that must be taken when handling twenty-one developing negatives..

After the plates and films are developed, I make it a practice to examine and study all of the negatives of the case in hand for any errors of technique, movement or change of position of the patient, also to detect any pathological or physiological variations that have not been clearly shown. During the hour which is allowed to each full-denture case, this is thoroughly attended to while the patient waits, and the areas are re-radiographed, if necessary, before the patient leaves the office. To prevent fading, the negatives should then be washed in clear, running water for from one-half to three quarters of an hour to remove all traces of "hypo." The next day, when the intra-oral negatives are dry, they are mounted between two plates of glass in their anatomical order and in the form of the arch. They are pasted onto the glass with passepartout binding, and marked, as to location, with a serial number for later reference and with the date of making. The top glass is then put on and the edges sealed. Thus, a permanent well protected record is made. The extra-oral plates are bound, numbered and dated.

One other problem should be discussed before proceeding. Patients are invariably inclined to ask the radiographer about the pathological conditions present, if any, shown by the radiograms. At first, it was difficult to refrain from answering this question because the fee

had to be collected and the patients felt that they should get some information before paying. We make it a practice to accept no patients unless they have been referred by a physician or a dentist, and then it is understood that the finished radiograms are to be delivered to the doctor who referred the case. My secretary attends to all financial arrangements with the patients. Thus, under present conditions, the question as to what the findings are is seldom presented to me. But, when it does arise, the patients are informed that the doctor who referred the case will discuss the diagnosis with them. However, a report of the radiographic findings, which is in no sense a diagnosis, is rendered only upon the request of the referring clinician.

III. DENTAL CONDITIONS THAT MAY BE RECOGNIZED IN, AND CORRECTLY INTERPRETED FROM, ACCURATE RADIOGRAMS, AS VERIFIED CLINICALLY OR EXPERIMENTALLY

For what purpose does the dentist send the patient to the radiographer when he asks to have only a few teeth radiographed? Each branch of dentistry has certain interests of major importance on which the radiogram throws a flood of light. The general practitioner looks for pulpless teeth, to see whether radiolucent areas are shown at any of the apices and whether the canals in such teeth are filled to the apices; also, to note the length and degree of curvature, if any, of the roots, should the canals need retreatment or refilling. The pyorrhea specialist wishes, in addition to this, to see how deep are the pockets around the loosest teeth. The extraction specialist desires, possibly, to see the position of an impacted tooth, signs of radiolucency around the apex of a tooth, or the location of a fractured root. The orthodontist seeks to locate unerupted teeth or teeth in malposition.

Let us assume that each notes the existence of conditions for which he seeks. But what, among other pathological conditions disclosed by the accurately made radiogram, has he overlooked? Here we come to the matter of appreciation of the higher values of the radiogram.

As I understand dentistry, it is a profession that is devoted to keeping healthy and efficient the organs of mastication. Why, then, do many practitioners give their attention, when one or more of the

normal array of thirty-two teeth is plainly diseased, exclusively to the teeth that are seen to be diseased and assume that the other teeth are above suspicion? Because, it appears, many dentists do not approach their patients with a view to examination of the mouth as a whole, with all its parts closely interrelated and interdependent in health as well as in disease, but confine themselves to the parts that show unmistakable signs of disease or abnormality. This attitude has not been entirely their fault. Dentists, prior to the advent of radiography, did not have a method of getting a "broad" view of the mouth as a whole, with its obscure conditions made almost as plain as its obvious conditions. And even after the advent of radiography, the radiogram was deficient until oral radiography attained the scientific accuracy that may now be claimed for it.

It is only by means of accurately made radiograms of the entire denture that the dentist can ascertain all the facts he should seek. I contend—not because I am a radiographer but because years of experience, and of close association with clinicians, have convinced me of the soundness of my view—that dentistry cannot achieve its best results without routine use of full-denture radiograms. Dr. Sumner Hardy was the first dentist in general practice, I believe, who insisted upon having full-denture radiograms of each of his new patients, before he attempted to make a diagnosis. Dr. Hardy's practice, in this relation, is now being followed by many others, but not as generally as it deserves to be.

Without an understanding of the normal, the abnormal cannot be understood. The normal condition of bone in each patient must be known before we can recognize what is abnormal for bone in that patient. To arrive at such an understanding, one must have full-denture radiograms of the patient. This is especially desirable with cases in which there is a possibility of systemic involvement.

Through the agency of extensive research, conducted to supplement our radiographic studies, we have been able to test our actual radiographic findings in detail and to establish a reliable foundation upon which the dentist may base his interpretation of radiograms. This research has been continued for so long a period, it has been checked and rechecked so often, and similar conditions have been shown to disclose themselves so regularly in the same way upon our

radiograms, that concise definitions of conditions shown in clear and accurate radiograms can now be made with complete confidence that these definitions are reliable in detail.

The conditions classified below have been recognized and correctly interpreted by the comparatively few clinicians who, having the advantage of accurately made radiograms, have verified their radiographic interpretations, "in the surgery," through operations by the Novitzky method (4, 5, 6, 7); or, have witnessed such operations. Heretofore, no effort has been made to classify conditions specifically in terms of their radiographic appearance, or to establish a definite terminology. This we are herewith attempting to do, at least provisionally, on the ground that, until such a terminology is agreed upon, there can be no satisfactory basis for clear discussion.

The names used to signify specific conditions have been adopted as the only possible alternative to coining an entirely new terminology. While some of these terms may be inaccurate, to a certain extent, there is ample precedent, nevertheless, for their use in denoting certain physiological and pathological changes which are known to both the medical and dental professions. For definitions, we have followed standard medical dictionaries.

My dental collaborators in the work of preparing this classification—upon whose judgment in unanimous agreement I rely entirely for the dental opinions expressed here with their approval—are Drs. William A. Colburn, Henley E. Miller, Joseph J. Pfister, Sumner Hardy, Harry A. Tuckey, Theodore C. Muegge, Shirley J. Ashby, Frank H. McKevitt, and Robert Burns, Jr., of San Francisco.

The "oral radiographic appearances" mentioned in the appended classified summary are shown in detail in *plates 1* to *9*, inclusive (pages 491–509), where the illustrations are marked at the significant places and the accompanying legends give additional suggestions. The illustrations in the nine plates correspond, respectively, with "radiographic appearances" for the nine successive classes specified below.

Class 1.—Periodontitis: Inflammation of the periodontal membrane

Oral radiographic appearance. Separation between the periodontal lamella and tooth (8); shown as a dark line around the tooth of greater width than that around the normal teeth.

Remarks. Probably the earliest stage that may be shown of inflammatory changes either in the tooth or surrounding alveolus. May be of infective or traumatic origin.

Oral operative findings. At operation this condition is evidenced by congestion of the periodontal membrane, which is discolored and on digital examination has a cushion-like feeling. The apex of the tooth feels smooth. The wall of the alveolus or periodontal lamella is hard and smooth with no apparent perforation.

Class 2.—Alveolitis: Inflammation of the alveolus or osseous wall of the tooth socket

Oral radiographic appearance. Thickening of the periodontal lamella, or obliteration of the periodontal lamella, and a merging of shadows cast by the tooth and the alveolus; or as a slight recession of the interdental alveolar crest.

Remarks. Periodontal irritation may be causing changes in the alveolus. May be of infective or traumatic origin.

Oral operative findings. At operation the periodontal membrane is found to be slightly congested at the middle- and apical-thirds, with marked congestion at the apex of the tooth, which is roughened. The periodontal lamella may be perforated. When this occurs, the cancellous process beyond the lamella is soft. Other types of alveolitis show the root to be hypercementosed and the surrounding process to be sclerosed.

Class 3.—Granuloma: A tumor-like mass, found at the apices of certain teeth and in some edentulous areas

Oral radiographic appearance. A cavity shown in bone, with a definite or slightly diffused outer wall. This bony wall is clearly defined, if there has been no breaking down of the granuloma. Appears as a dark cavity with a more or less dense border.

Remarks. Probably a proliferation of the periodontal membrane, with absorption of bone due to the pressure caused by the expansion of the enlarging mass. May be of infective, traumatic, or medicinal origin.

Oral operative findings. At operation it appears as a red, spongy, tumor-like mass attached to the tooth and lying in a small cavity in the process, but by removing the tooth and the attached granuloma, it may be found that the granuloma is more firmly attached to its osseous surrounding wall than it was to the tooth. The cavity in which the granuloma lay is found to have a fairly well-defined but irregular wall.

Class 4.—Cyst. A cavity, in the cancellous bone of a jaw, that contains fluid and that has a limiting membrane (2)

Oral radiographic appearance. A non-osseous cavity in bone with a very definite limiting border and a sharply defined bony outer wall or table. Appears as a large cavity in the bone with a clean cut, dense, outer border.

Remarks. The extensions of cysts often involve adjacent parts. Such possible involvement should be carefully studied in a number of radiograms from various angles.

Oral operative findings. At operation it appears as a soft, rounded, mass with a pale capsular membrane. Upon retracting the periosteum, it may be found that the labial, buccal, or lingual plate has been entirely absorbed and the cystic membrane has become adherent to the periosteum. If the capsular membrane is ruptured, the mass collapses, and a serous exudate appears. Some of these cysts may be removed en masse. The bony wall is smooth and lined with a silk-like membrane.

Class 5.—Pyorrhea (alveolaris): Progressive necrosis of the dental alveolus and investing tissue

Oral radiographic appearance. The various stages of this disease are indicated by the amount of recession, from normal, of the alveolar process at the necks of the teeth. Both labial and lingual plates can be shown. The process presents serrated edges, and the gingiva casts shadows, in radiograms, if there is much congestion in the soft tissues. Calculus (on the tooth surfaces) is shown whenever it occurs.

Remarks. Periodontitis is probably the first stage of this disease that shows in a radiogram. Malocclusion, infra-occlusion, trauma, overhanging fillings, crowns that do not fit, loss of contact points, and unsupported teeth, are signs for prognosis in this condition. Alveolitis may be the second stage shown in a radiogram.

Oral operative findings. On making the sub-periosteal incision and retracting the gingival tissues, the teeth appear to be eroded and may be very loose, the alveolus is soft and necrotic, the border is irregular and roughened. Attached to the periosteum, and surrounding the roots of the teeth and alveolus, are found masses of disorganized and cystic material, which is very vascular and hemorrhagic (3).

Class 6.—Necrosis (alveolar): Disorganization and death of portions of cancellous bone in the jaws

Oral radiographic appearance. This type of diseased bone has irregular ill-defined outlines and a foggy mottled structural appearance of the cancellations; and has a lower radiographic density than normal bone. The gingival border is serrated and irregular.

Remarks. Generally found at sites of old extractions or at the apices of some pulpless teeth. The pathological conditions remain after extractions. This type of bony condition changes rapidly by absorption. There is no visible external drainage, or there may be drainage into soft parts, or infiltration into an antrum or inferior dental canal.

Oral operative findings. After the sub-periosteal incision is made and the gingival tissues are ready to be retracted, it is often found that the periosteum is very difficult to retract without trauma when it invests an underlying necrosis. After retraction of periosteum, the labial plate may be found to be very compact. When the plate is punctured, a thin watery exudate is discharged. The process appears to be inflamed and soft. The labial plate, in some types of necrosis, has entirely disappeared and is invested only by periosteum. The cancellous condition described above is similar in both types but there are some variations. Both are very vascular and hemorrhagic until all the pathological tissue is removed and healthy bone is reached. The necrosis is usually more extensive than the radiogram indicates, because the necrosis frequently extends toward the lingual plate.

Class 7.—Osteosclerosis: A condition in which portions of bone become dense and hard

Oral radiographic appearance. Very dense mass in bone or at the apex of a tooth. Has an irregular but well-defined outline and no cancellations are shown. Often is surrounded by a mass of much lower density.

Remarks. Found at the sites of old extractions; at the apices of some pulpless teeth, as well as of teeth that are not pulpless but have or have had pulpitis; around foreign bodies or fragments of fractured roots; at points of pressure either from malocclusion or trauma. May be of infective origin or arise from trauma. Especially important in neurological cases.

Oral operative findings. At operation it is found to be a hard ivory-like process that resists the chisel or burr. Is irregular in outline and is difficult to distinguish except by the sense of touch. It is not removed, as a rule, unless it presses on a nerve trunk or is surrounded by an area of very soft bone.

Class 8.—Osteitis serrata: Sharp saw-edged bone on the ridges of some edentulous jaws (3)

Oral radiographic appearance. Shown as a thin ridge of serrated bone, 1 to 4 mm. above the general outline of the process. The gingival tissue often casts a well-defined shadow, showing congestion over this type of bony condition.

Remarks. This type of bony abnormality often occurs in edentulous portions of the jaws, and produces a condition on which it is almost impossible to maintain, comfortably, dentures or saddle bridges.

Oral operative findings. At operation it is shown as an irregular, hard, sharp ridge of bone, with zones or portions that are soft. The sharp projections may extend in one or three directions. Small portions of this bone may be very vascular and hemorrhagic. This may also be said of the investing gingival tissues, which usually show hypertrophy and signs of irritation.

Class 9.—Osteogenesis: Healthy bone regenerated where a tooth had been extracted, or where pathological conditions had existed and were eradicated

Oral radiographic appearance. Has slightly greater density than normal bone, but the cancellations are uniformly irregular, clean cut, well defined, and appear to be healthy. The gingival border is a selvage.

Remarks. Models, cultures, and radiograms, of this type of bone have shown that, in such cases, no absorption has taken place in a period of over two years. Dentures may be made that can be regarded as fairly permanent.

Oral operative findings. Only four cases of this type of bone have been entered surgically, then only for scientific reasons and to get cultures. The periosteum retracted easily, the labial plate was firm and healthy, and hemorrhage occurred after puncturing the plate. The cancellations were hard and firm, and the cultures were sterile.

IV. CONCLUDING REMARKS

The evidence gathered, so far, from a bacteriological standpoint, tends to demonstrate that organisms are harbored by some of the areas and structures investigated and classified by us. We are not in a position to present the details of the findings, because we have not yet obtained enough data to justify conclusive statements

concerning the bacteria that appear in the respective structures examined. A detailed report will be published in the near future, which will supplement the foregoing classification and interpretations, and will indicate the significance of the material taken from the tissues in question.

Sufficient data have been presented in this paper, to show, we believe, that great advances have been made recently in oral radiography. It should be plain to everyone that these advances have been made under the compulsion of a clear idea of the objective of oral radiography. What is that objective? It is not a selfish one; selfish, I mean, in the sense that its sole aim is to benefit radiography. Radiography, of course, is a supplemental science. Greater perfection in it is particularly valuable because that will mean the attainment of more perfect dental and medical work. That, of course, is the objective of the dental or medical radiographer. We aim to be a better-working tool for the professions whose activities we supplement. But we expect those professions to realize the tremendous value of that tool, and to use it.

We count it one of the achievements of radiography that it has pointed out a path along which the medical and dental professions can advance together. Along that route real progress is going to be made. When the close interrelation of oral conditions with those in widely separated parts of the human system are recognized as universally as the facts of secondary focal infections seem to warrant, then, indeed, great progress will have been made by both professions.

The basis of such progress, I believe, is scientific radiography. When the dental profession fully realizes the value of accurately made radiograms, and insists upon getting such radiograms, and then uses them for every particle of value they possess, the dental profession will be on the direct road to that high destiny of helpfulness to humanity for which it is clearly destined.

209 Post Street

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PLATES 1–9

(McCormack: Standardized Oral Radiography)

PLATE 1

Periodontitis

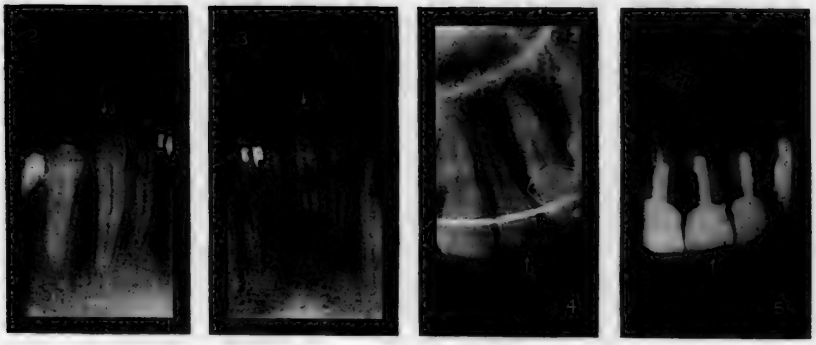
1. Left mandible: teeth not in occlusion. *X*.—Lower first molar: caries under the filling, which may involve the distal horn of the pulp. *Periodontitis is shown as a dark line around the apices*. *Y*.—Lower second molar: periodontitis, probably from traumatic occlusion. *Z*.—Lower third molar: dark area around apices, probably from superimposition of the overlying muscular tissues.

2. Lower right cuspid. The dark line extends up to the gingival border. Lack of contact and trauma have probably produced the periodontitis.

3. Lower left central. The process has receded from around the neck of the tooth. The dark line of periodontitis extends almost around the tooth surface. The apex is slightly denuded. The contacts are good. Probably gingivitis.

4. Upper left lateral. The dark line extends completely around the root. There is a marked thickening of the periodontal lamella, probably traumatic.

5. Upper left central. Pulpless, with a partial root-canal filling, post and crown, and caries under the crown. The dark line around the entire root shows periodontitis.



(McCormack: Standardized Oral Radiography)

PLATE 2

Alveolitis

6. Left mandible: upper and lower teeth in occlusion. Lower left first molar: extensive caries, probably involving the pulp chamber. Periodontitis is shown around the apices of the roots. Surrounding this are other areas of lower density, which show an alveolar change or alveolitis.

7. Upper left first bicuspid. Pulpless tooth with partial root-canal filling. There is an increased thickening of the process surrounding the apex.

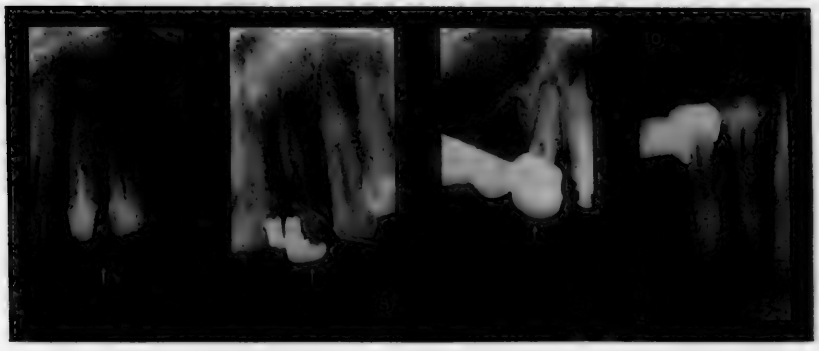
8. Upper left lateral. Deep filling probably involving pulp canal, which shows a degenerative change. There is a separation between the apex and the thickened periodontal lamella, which probably denotes pulpitis.

9. Upper right first bicuspid. Pulpless, with a wire in one canal; the other, unfilled. Periodontitis and alveolitis are shown around the entire root.

10. Lower right first bicuspid. Vital tooth; periodontitis is shown on the distal surface and extensive recession of the alveolar process is shown on the mesial surface.

11. Lower left first molar. Pulpless, with partial root-canal fillings. The mesial root shows periodontitis extending upward to about the apical third and a very thickened periodontal lamella around the apex. The distal root shows a thickened lamella and a slight thinning of the process at the apex.

12. Lower right second molar. Pulpless tooth that casts very little shadow and appears to have merged into the surrounding process. Many such teeth, after extraction, are found to be very brittle and fracture easily.



(McCormack: Standardized Oral Radiography)

PLATE 3

Granuloma

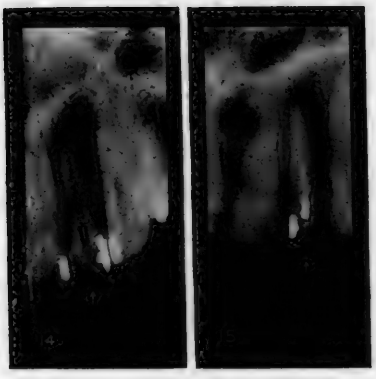
13. Lower left mandible: not in occlusion. Lower first molar: deep fillings, probably involving the pulp chamber. Periodontitis is shown on the root surfaces. Small but well defined granulomata are shown at both apices.

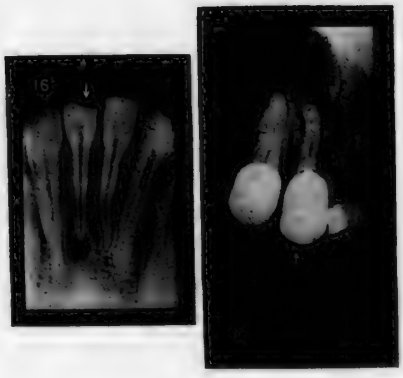
14. Upper left lateral. The pulp canal in this tooth has not been entered, but there is an extensive granuloma around the apex and an area of much greater extent around the granuloma, which may be the beginning of necrosis.

15. Upper left lateral. Pulpless tooth, with the root-canal filled to the apex and a small granuloma around the apex.

16. Lower right central. Apparently a traumatically devitalized pulp, as no opening into the pulp canal is shown. A marked granuloma is shown at the apex, which is slightly denuded.

17. Upper right first bicuspid. Pulpless tooth with a partial root-canal filling and a crown. An apical granuloma is shown superimposed over the antrum of Highmore.





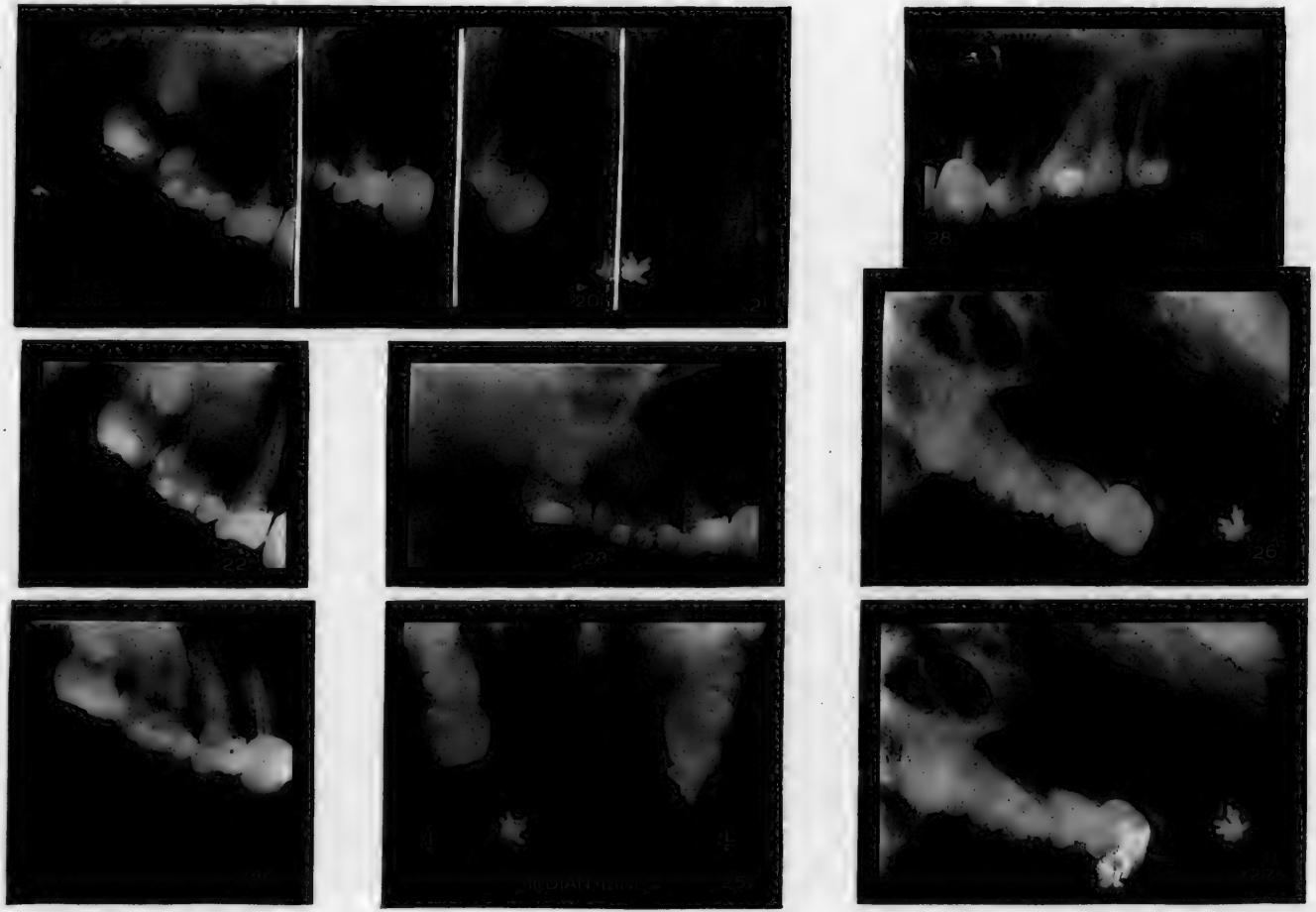
(McCormack: Standardized Oral Radiography)

PLATE 4

Cyst

18 to 27, inclusive. Upper right central to upper right second molar: radiogram made from various angles to show the extension of the cystic condition. A large cyst may be seen in the maxilla involving the apices of all these teeth as well as a considerable portion of the roots. The cyst extends to the antrum, the floor of which, together with the palatine plate, is probably involved.

28. Upper left bicuspid. Pulpless tooth, crowned, with a partial root-canal filling; has a small apical cyst.

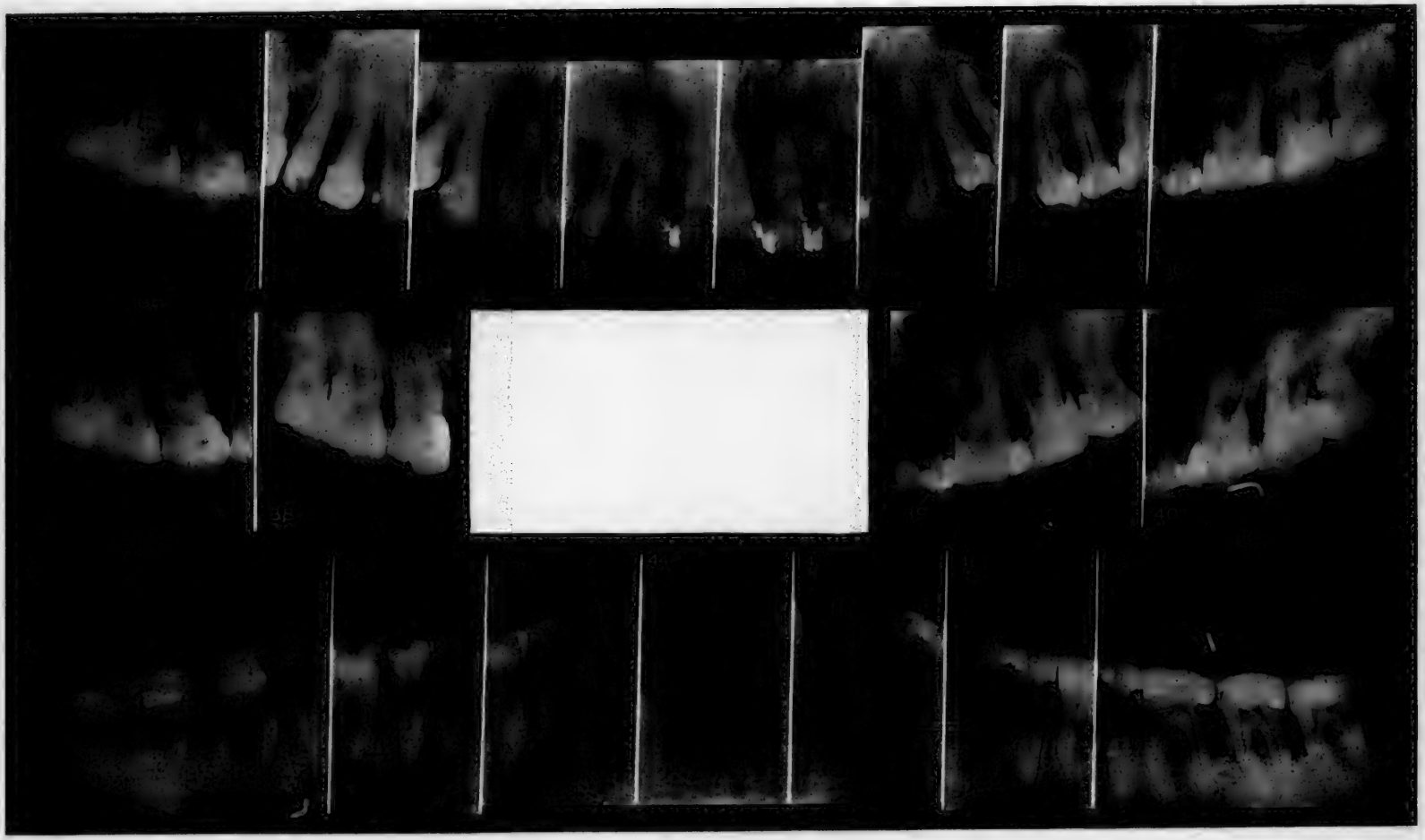


(McCormack: Standardized Oral Radiography)

PLATE 5

PYORRHEA

29 to 47, inclusive. Entire denture: shows extensive recession of the alveolar process, probably due to pyorrhea alveolaris. The radiograms of the upper molars were made from various angles to show the amount of recession, if any, in the bifurcations.



(McCormack: Standardized Oral Radiography)

PLATE 6

Necrosis

48. Left mandible: teeth in occlusion. Lower right first molar: crowned and pulpless, with a partial root-canal filling. The dark shadow around the roots of this tooth, and extending to and around the mesial root of the second molar, shows necrosis. (There is a mass of root-canal filling material in the bifurcation of the roots of the first molar.)

49. Upper left bicuspid area; edentulous. Irregular, ill defined outlines. There is a foggy, mottled, structural appearance of the cancellations, and the borders of this condition appear to shade off into the normal bone.

50. Upper left cuspid and first bicuspid region; edentulous. Necrosis surrounding a remaining granuloma, which was probably left at the time of extraction. The condition extends almost to the floor of the antrum, which appears intact.

51. Lower right bicuspid and first molar region. The edentulous area under the bridge looks like that described for tooth 49, except that the overlying gum tissue shows a marked thickening.

(McCormack: Standardized Oral Radiography)

PLATE 7

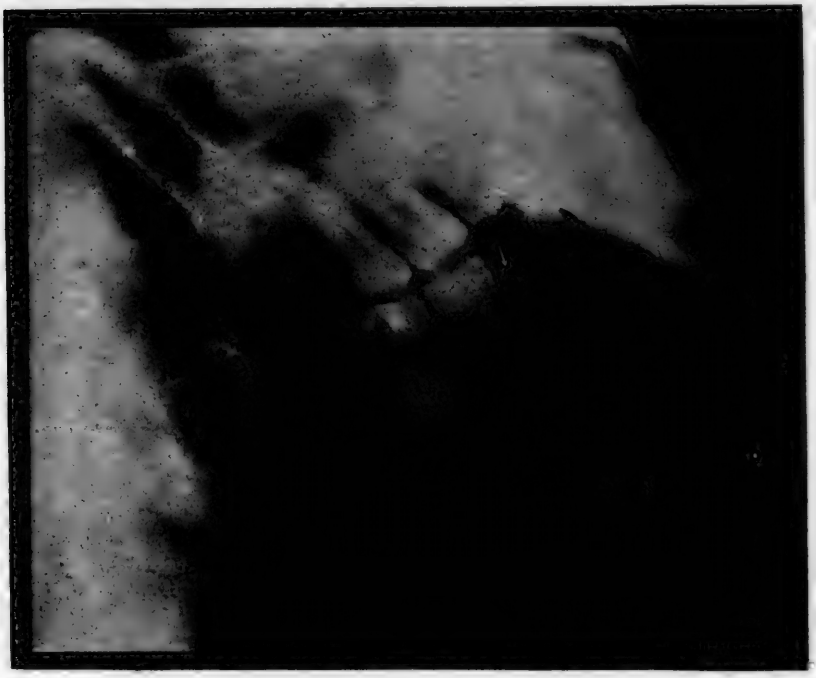
Osteosclerosis

52. Right mandible: teeth in occlusion. Lower left second bicuspid area; edentulous from recent extraction. There is alveolitis and sclerosis, which extend down to and around the inferior dental canal, constricting the canal.

53. Lower right first molar. Apparently vital, but with a deep filling, caries under the filling, and pulp encroachment (possible pulpitis). Sclerosis extends from the apices downward to the inferior dental canal, which appears to limit the canal inferiorly.

54. Lower left first molar area; edentulous. Small piece of foreign body surrounded by sclerotic wall.

55. Lower right second and third molars; vital. There is an area of sclerosis surrounding both teeth, rendering it difficult to discern the roots of the third molar. (Traumatic sclerosis, probably from malocclusion).



(McCormack: Standardized Oral Radiography)

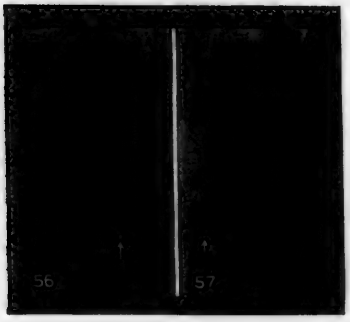
PLATE 8

Osteitis Serrata

56 and 57. Upper incisor area; edentulous. Irregular, "saw edge," sharp spicules of bone extending downward from the maxillary process.

58 and 59. Lower incisor area; edentulous. Shows conditions similar to those for teeth 56 and 57, together with a hypertrophied gum.

60 and 61. Lower left incisor area; edentulous. Sharp spicules and filaments of the process with a razor-like edge.

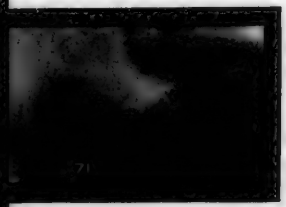


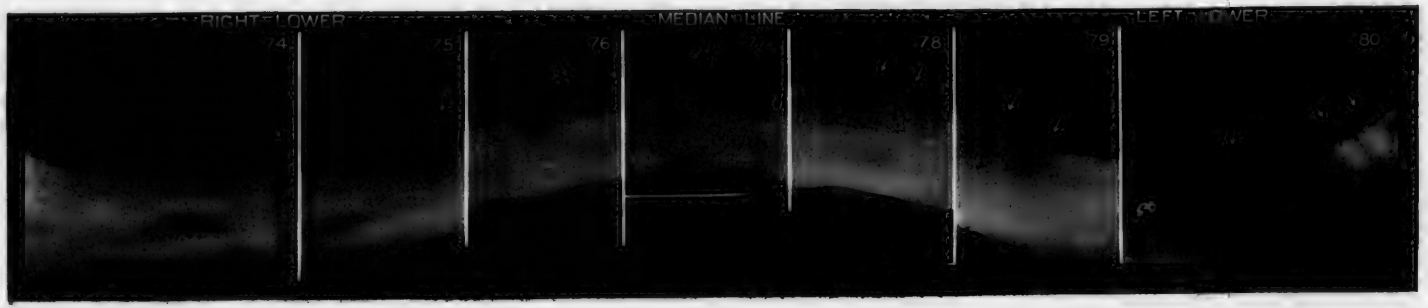
(McCormack: Standardized Oral Radiography)

PLATE 9

Osteogenesis

62 to 80, inclusive. Entire mouth; edentulous. Normal regeneration is shown, except where root fragments were left unextracted (in 62, 63, 64, 80) and where the arrows indicate small areas of local necrosis (in 67, 68, 69, 76, 77, 78, 79). In 74, 75 and 80, there are small areas of osteitis serrata, with sharp thin spicules of the process extending outward.





(McCormack: Standardized Oral Radiography)

EXPERIMENTAL STUDIES OF THE VALIDITY OF ADVERTISED CLAIMS FOR PRODUCTS OF PUBLIC IMPORTANCE IN RELATION TO ORAL HYGIENE OR DENTAL THERAPEUTICS

6. May the Use of Pebeco Tooth-Paste, Twice A Day, Be Reasonably Regarded as a Part of "the Basis of Correct Oral Hygiene?"

PART II. TESTS OF THE RELATIVE TOXICITY OF POTASSIUM CHLORATE AFTER INTRAVENOUS INJECTION INTO ALBINO RATS

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I. INTRODUCTION

The manufacturers of Pebeco Tooth-Paste have advertised the claim that the use of that paste as a dentifrice, "twice a day," is a part of "the basis of correct oral hygiene" (3). Distinguished physicians and dentists have denied the validity of this claim in behalf of the use of Pebeco as a dentifrice, on the ground that potassium chlorate (present in Pebeco to the amount of 50 per cent) is "an extremely dangerous drug, death often resulting from its unguarded use," and that "grave perils attend the employment" of dentifrices, such as Pebeco, that contain potassium chlorate in large proportions (3).

It is obvious that, if potassium chlorate is an "extremely dangerous drug" and if Pebeco Tooth-Paste contains 50 per cent of it, the denti-

fricial use of Pebeco, "twice a day," cannot be truthfully advertised as a part of "*the* basis of *correct* oral *hygiene*."

In an effort independently to ascertain the facts in this situation, and to do so on the basis of experimental findings, I have been conducting an investigation of the toxicity of potassium chlorate, determined directly and in comparison with the toxicity of various familiar substances of either physiologic or toxicologic import, or both, such as sodium chloride, potassium sulfocyanate, and mercuric chloride. *Part I* of this study presented the results of "preliminary tests of the relative toxicity of potassium chlorate for tadpoles and newts" (1). In the tests there described, the animals were kept continuously immersed in solutions of potassium chlorate or of substances with which the toxicity of potassium chlorate was compared. It was concluded, judging from the mortality records of the tests, that "potassium chlorate was somewhat more toxic than common salt, but much less poisonous than common soaps or sodium arsenite, and very much less destructive than mercuric chloride; it was also less damaging than potassium sulfocyanate."

These results for tadpoles and newts cannot be arbitrarily applied to human conditions. The results do indicate, however, that, for man, potassium chlorate is not as "*dangerously*" poisonous, and that its dentifricial use is not as "*gravely*" perilous, as has been believed; but, of course, these findings do not show that potassium chlorate is not a poison or that potassium chlorate may be taken into the body with impunity. The results suggest, and prepare the way for, an extension of the experiments.

In this, the second, part of this investigation, an effort was made to obtain results for the absolute and relative toxicities of potassium chlorate that would be more sharply defined mathematically than those of Part I; that would show effects after direct introduction into the blood; and that would relate to a type of animal more nearly allied to man. Accordingly, solutions of potassium chlorate and comparable substances were injected intravenously into a hundred albino rats, under conditions that have yielded results of special value and significance.

II. EXPERIMENTAL (PLAN, METHODS)

Plan

Part I of this series of studies was directed primarily at the question of the *absorbability* of potassium chlorate (1). It was said in this relation (page 315): "One of the most important considerations involved in the toxicology of potassium chlorate, as an ingredient of dentifrices, is the question of its *absorbability through the oral mucous membranes*. Does potassium chlorate, introduced into the mouth in the form, say, of Pebeco, pass into and through the oral mucous membranes in sufficient quantity, at a time or in time, to damage the membranes (and injure the person)? This question presents the crux of the whole problem under review." It was believed, at the outset, that the results of comparative tests of the toxicity of potassium chlorate and other substances, on tadpoles and newts continuously immersed in solutions of these substances, would show clearly that potassium chlorate is or is not readily absorbable into the blood through membranes similar to those in the human mouth, and that potassium chlorate is or is not an "*extremely*" poisonous substance after its passage by absorption into the blood. The findings indicate, in a general way, that although potassium chlorate is readily absorbable and also toxic, it is not, as has already been said, as dangerously poisonous as some believe it to be (3).

Toxicologic results for a given species may not apply with equal significance to other species. A fair objection to the application of the results obtained with tadpoles and newts, to *human* conditions, is the *possibility* that tadpoles and newts are relatively less susceptible than men to the action of potassium chlorate. (Of course, on the other hand, they may be more so.) It is *possible* that these cold-blooded animals are materially different from men in the comparative degrees, conditions, and rates, of their absorption of potassium chlorate, even under the most favorable conditions for its transmission to the blood, and that, therefore, the results for tadpoles and newts mean less (or more, of course), for men, than they seem. It is *conceivable*, also, that such cold-blooded animals do not show the *degree* of injury they would if potassium chlorate were absorbed into their blood (in such experiments as those described in Part I), in the man-

ner and to the extent that it is absorbed in men (presumed here for the sake of judicial argument), *from the mouth*, or stomach, or both, when Pebeco and similar products are used as dentifrices.

In order to resolve these doubts, however extreme they may be, and also to extend the scope and deepen the import of this investigation, (a) the question of *absorbability* into the blood was entirely removed from consideration, for the time being, by the simple expedient of injecting solutions of the compared substances *directly into the circulating blood itself*, (b) and a higher type of animal—the albino rat—used for the purpose. This animal, an omnivorous mammal, unlike the tadpole or newt, closely resembles man in dietary habits, nutritive character, physiological quality, and toxicologic responsiveness; and affords a more conventional basis for the determination of toxicologic properties that are applicable to, and demonstrable in, man.

Experiments on men will be postponed until it is certain that potassium chlorate is either so poisonous that such experiments would be unjustifiable, in which event they would not be attempted; or so innocuous, that adequate experiments in this relation would not be dangerous in any degree and could be conducted with entire fairness to the individuals involved.

The literature on the toxicology of potassium chlorate will be reviewed with the discussion of a later series of findings, when all the available facts can be summarized to the greatest advantage.

Methods

Animals. The animals used in these tests were selected from our own colony of albino rats, which has been in process of *continuous* development in this laboratory for a period of seven years. The colony has been developed for the routine and experimental work of this laboratory. Only the healthiest adults have been used for breeding purposes and abnormal rats of all ages have been promptly chloroformed. As a consequence (the animals having been given an abundance of good food and fresh water daily, and confined in suitable cages that have been cleaned once a day), the available rats in the colony are notable for their uniform vigor and outstanding normality—"survivals of the fittest" as well as of the most suitable for such experiments as those described below.

Diet. From the beginning of their independent nutritive existence, the rats were fed daily an abundance of good "balanced" food, on the following weekly dietary schedule: *Sunday*—fresh cabbage, cheese; *Monday*—cracked corn, beef-heart; *Tuesday*—milk, wheat bread; *Wednesday*—fresh cabbage, beef-heart; *Thursday*—fresh turnip, cheese; *Friday*—rye grain, wheat bread; *Saturday*—oats, beef-heart. The rats were also fed on this dietary schedule immediately after a series of injections was completed, and thereafter to the end of the period of observation.

Chemicals employed. The best available chemical preparations were used in the injections. With the exception of the soaps (see foot-note in *table 1*), all the products were Eimer and Amend "*tested purity*," or Kahlbaum "*K*," preparations. The portions of the products intended for the solutions to be used in the injections were taken from previously unopened containers, and were weighed directly as removed therefrom. No attempt was made to eliminate atmospheric moisture from the mass of the product to be weighed, but all due care was taken to prevent introduction of moisture and impurities. All the products were dry; and any error due to original inclusion of atmospheric moisture, in the weights of products taken, must certainly have been within the range of influence of the unpreventable variations in the animals themselves—variations without material bearing on the significance of results ("average") obtained for a relatively large number of animals, as was the case for each substance tested in this work. The solutions prepared for the injections (4 to 16 per cent) were made in volumes of 100 cc.

Injection technic. The injection procedure was identical in principle, and in most details, with that required by the U. S. Government for official tests, on albino rats, of the toxicity of arsphenamine (Salvarsan). It was modified in non-essentials to meet certain desirabilities in these tests and conducted as follows:

From twelve to fifteen hours prior to the times at which the individual injections were to be made (the requirement in the official method above referred to is twelve to eighteen hours), the well nourished rats selected for a given test were removed from cages containing an abundant supply of food and water to one containing no food but an abundance of water. (This precaution prevents disturbing variations in weight, and consequent errors in the calculations of dosage for the subsequent injections, that might otherwise be introduced by inequalities in the amounts of food in the alimentary tracts of the animals involved.) Just before a series of injections was started the animals to be treated were carefully weighed in grams, the weights recorded in the nearest whole numbers (maximum deviation, 0.5 gram), and the rats put temporarily into small individual cages. (Neither food nor water was given during this short period.)

One rat after another was then fastened, outstretched on its back, on an ordinary holder (not too tightly to interfere with respiration); the hair removed from the inside surface of the upper right hind leg, with a barium sulfid-soap-flour-talcum-water mixture; the saphenous vein exposed by a suitable superficial incision (without the aid of an anesthetic, as required in the official method mentioned above); and the calculated volume of solution then carefully and visibly injected, in toto, into that vein with a minutely and accurately calibrated Luer syringe, with a very fine needle, from which deliveries of 0.01 cc. could be made completely and continuously under control. The injections were made as closely and as uniformly as possible at a rate of 0.1 cc. of solution in 15 seconds, the rate rarely becoming more rapid than 13 seconds nor slower than 17 seconds, and averaging 15 seconds, per 0.1 cc. of solution.

Immediately after the completion of the injection in each case, the wound was closed with a single stitch of silk thread, and the rat isolated in a small cage until the remaining injections were concluded. In every instance in which an animal survived the injection, the wound over the saphenous vein healed rapidly and uneventfully.

Hemorrhage from the vein and wound was always insignificant—frequently it did not occur.

Period of observation. The surviving animals for a given test, after being suitably marked for individual identification, were assembled in one cage and kept there until the end of the period of observation—at least two weeks in every instance. With them in the same or near-by cages, "control" (untreated) animals of the same age, size, and sex were closely associated, to afford direct comparisons of treated with normal untreated animals, and thus to guard against misinterpretations of any unexpected or unaccountable deviations in condition (none of which arose).

The tests were conducted, from time to time, during the months of June, July and August.

III. SUMMARY OF THE RESULTS

Tabulated data. The mortality record and further details of the tests are shown in *table 1*.

Table 1 gives the essential data for each substance named in the sectional headings (I–VII). For each group of tests, in sub-sections A to V, is stated (a) the kind of product employed in the corresponding test; (b) the concentration of the solution injected; (c) the sex of the

individual rats; (d) the length of the period immediately preceding the injections during which the previously well-fed animals were continuously deprived of food (with water always available); and (e) the added length of the injection period during which neither food nor water was received by the rats prior to their replacement in cages where food for the day, and water, were freely available. (The total amount of time involved in conditions "d" and "e," above, is the length of the period during which no food had been received by the rat *last* treated.)

The columns in *table 1*, as shown by the general headings, give (a) the sequence numerals of record for the individual rats; (b) the weights of the rats in grams taken a few minutes before the beginning of the injection period for the corresponding groups; (c) the volumes of solution injected into the individual rats, expressed in tenths of cubic centimeters ("cc./10"); (d) the actual amounts of the tested substances contained in the injected volumes ("total dose"), expressed in milligrams; (e) the amounts of the substances thus injected, calculated per kilo of the body-weights of the corresponding rats ("dose per kilo"), expressed in milligrams; and (f) indications of the toxic results, after the injections, expressed to indicate survival ("+") to the end of the *shortest* period of observation (two weeks), or death ("−") *within* the number of minutes, after the *completion* of the injections, specified by the corresponding numerals.

Miscellaneous additional data. Each group of treated animals that survived, and all the control rats, weighed more at the end of the period of observation than they did at the time the corresponding injections were made. As this general statement presents the essential facts in this relation, all the weights for control animals, and for the groups of treated animals at the ends of the tests, are omitted from the printed record, as non-significant.

All the animals that survived were lively and vigorous at the ends of the periods of observation, a fact showing clearly that the large tolerated doses did not exercise cumulative or continuing toxic effects.

The toxic symptoms, in the tests that terminated fatally, were such as are characteristic of the substances injected. Death through the action of the largest doses of *sodium chloride* and *sodium chlorate* was attended by convulsions, and by edema of the lungs with extrusion of froth from the nostrils. Cyanosis to a marked degree

TABLE 1

Data pertaining to the comparative toxicity of potassium chlorate, sodium chloride, potassium sulfocyanate, sodium nitrite, potassium chloride, sodium chlorate, and soaps (castile and sodium oleate), after their respective intravenous injection into albino rats

RAT		DATA PERTAINING TO THE INJECTIONS			RESULTS	
Number	Weight, grams	Volume injected, cc./10	Dose injected		Lived 2 weeks	Died: min.
			Total, mgm.	Per kilo, mgm.		
		I. Potassium chlorate				
A. Potassium chlorate (Eimer and Amend, "tested purity"): 4 per cent solution. Male rats. Food not available for 12 hr., 10 min., prior to the first injection. Length of the period during which the five injections were made: 52 min.						
1	189	12.2	48.8	258	−	4
2	176	10.6	42.4	241	+	
3	175	8.8	35.2	201	+	
4	172	11.0	44.0	256	+	
5	149	9.5	38.0	255	+	
B. Potassium chlorate (Eimer and Amend, "tested purity"): 4 per cent solution. Male rats. Food not available for 12 hr., 30 min., prior to the first injection. Length of the period during which the five injections were made: 58 min.						
6	222	14.4	57.6	260	+	
7	213	13.9	55.6	261	+	
8	212	14.9*			+	
9	206	14.4	57.6	280	+	
10	200	14.0	56.0	280	+	
C. Potassium chlorate (Kahlbaum, "K"): 4 per cent solution. Female rats. Food not available for 12 hr., 47 min., prior to the first injection. Length of the period during which the six injections were made: 1 hr., 8 min.						
11	100	7.0	28.0	280	−	1
12	101	7.1	28.4	281	−	1
13	107	7.0	28.0	262	−	2
14	108	6.8	27.2	252	+	
15	111	6.9	27.6	249	+	
16	128	8.0	32.0	250	+	
D. Potassium chlorate (Kahlbaum, "K"): 4 per cent solution. Male rats. Food not available for 12 hr., 15 min., prior to the first injection. Length of the period during which the five injections were made: 1 hr., 20 min.						
17	180	12.6	50.4	280	+	
18	198	14.4	57.6	291	+	
19	200	14.5	58.0	290	+	
20	223	16.0	64.0	287	+	
21	231	17.4	69.6	301	−	1

* Distilled water instead of potassium chlorate solution was injected into rat 8. There were no observable effects of either the water or the volume.

TABLE 1—*Continued*

Data pertaining to the comparative toxicity of potassium chlorate, sodium chloride, etc.

RAT		DATA PERTAINING TO THE INJECTIONS			RESULTS	
Number	Weight, grams	Volume injected, cc./10	Dose injected		Lived 2 weeks	Died: min.
			Total, mgm.	Per kilo, mgm.		
II. Sodium chloride						
E. Sodium chloride (Kahlbaum, "K"): 4 per cent solution. Male rats. Food not available for 12 hr., 35 min., prior to the first injection. Length of the period during which the six injections were made: 1 hr., 15 min.						
22	196	12.8	51.2	261	+	
23	189	13.3	53.2	281	+	
24	179	13.4	53.6	299	+	
25	178	15.1	60.4	339	+	
26	169	14.8	57.6	350	+	
27	165	14.9	59.6	361	+	
F. Sodium chloride (Kahlbaum, "K"): *8 per cent solution.* Female rats. Food not available for 13 hr., 15 min., prior to the first injection. Length of the period during which the five injections were made: 48 min.						
28	105	7.4	59.2	564	+	
29	101	8.1	64.8	642	+	
30	100	9.0	72.0	720	+	
31	95	10.5	84.0	884	+	
32	95	12.4	99.2	1,044	+	
G. Sodium chloride (Kahlbaum, "K"): *16 per cent solution.* Female rats. Food not available for 13 hr., 25 min., prior to the first injection. Length of the period during which the four injections were made: 40 min.						
33	103	8.3	132.8	1,289	+	
34	94	13.7	219.2	2,332	−	1
35	97	13.6	217.6	2,243	+	
36	151	22.7	363.2	2,405	−	8
III. Potassium sulfocyanate						
H. Potassium sulfocyanate (Eimer and Amend, "tested purity"): 4 per cent solution. Male rats. Food not available for 13 hr., 55 min., prior to the first injection. Length of the period during which the five injections were made: 58 min.						
37	196	4.7	18.8	96	+	
38	186	4.7	18.8	101	+	
39	186	5.6	22.4	120	+	
40	181	6.4	25.6	141	−	2
41	164	5.1	20.4	124	+	

TABLE 1—*Continued*

Data pertaining to the comparative toxicity of potassium chlorate, sodium chloride, etc.

RAT		DATA PERTAINING TO THE INJECTIONS			RESULTS	
Number	Weight, grams	Volume, injected, cc. /10	Dose injected		Lived 2 weeks	Died: min.
			Total, mgm.	Per kilo, mgm.		
III. Potassium sulfocyanate—Continued						
I. Potassium sulfocyanate (Kahlbaum, "K"): 4.0 per cent solution. Female rats. Food not available for 12 hr., 45 min., prior to the first injection. Length of the period during which the three injections were made: 34 min.						
42	109	3.3	13.2	121	+	
43	103	3.6	14.4	140	+	
44	101	3.8	15.2	150	−	1
IV. Sodium nitrite						
J. Sodium nitrite (Eimer and Amend, "tested purity"): 4 per cent solution. Male rats. Food not available for 12 hr., 30 min., prior to the first injection. Length of the period during which the five injections were made: 1 hr., 8 min.						
45	211	14.8	59.2	281	−	5
46	214	12.9	51.6	241	−	10
47	222	8.9	35.6	160	−	18
48	190	3.8	15.2	80	−	45
49	188	4.7	18.8	100	−	27
K. Sodium nitrite (Kahlbaum, "K"): 4.0 per cent solution. Male rats. Food not available for 13 hr., 55 min., prior to the first injection. Length of the period during which the four injections were made: 47 min.						
50	161	3.3	13.2	82	−	43
51	183	2.8	11.2	61	+	
52	193	2.0	8.0	41	+	
53	206	4.1	16.4	80	−	21
V. Potassium chloride						
L. Potassium chloride (Kahlbaum, "K"): 4.0 per cent solution. Male rats. Food not available for 12 hr., 30 min., prior to the first injection. Length of the period during which the six injections were made: 54 min.						
54	113	3.9	15.6	138	−	2
55	108	3.8	15.2	141	−	1
56	104	2.6	10.4	100	+	
57	101	3.0	12.0	119	+	
58	100	3.3	13.2	132	+	
59	99	3.2	12.8	129	+	

TABLE 1—*Continued*

Data pertaining to the comparative toxicity of potassium chlorate, sodium chloride, etc.

RAT		DATA PERTAINING TO THE INJECTIONS			RESULTS	
Number	Weight, grams	Volume, injected, cc./10	Dose injected		Lived 2 weeks	Died: min.
			Total, mgm.	Per kilo, mgm.		
V. Potassium chloride—Continued						
M. Potassium chloride (Kahlbaum, "K"): 4 per cent solution. Female rats. Food not available for 13 hr., 5 min., prior to the first injection. Length of the period during which the four injections were made: 46 min.						
60	204	6.6	26.4	129	+	
61	180	6.3	25.2	140	−	1
62	197	6.4	25.6	130	+	
63	175	6.1	24.4	139	−	2
VI. Sodium chlorate						
N. Sodium chlorate (Eimer and Amend, "tested purity"): 4 per cent solution. Female rats. Food not available for 12 hr., 40 min., prior to the first injection. Length of the period during which the five injections were made: 48 min.						
64	106	8.5	34.0	321	+	
65	104	10.4	41.6	400	+	
66	98	11.8	47.2	482	+	
67	93	13.0	52.0	559	+	
68	94	15.1	60.4	643	+	
O. Sodium chlorate (Eimer and Amend, "tested purity"): *8 per cent solution.* Female rats. Food not available for 12 hr., 35 min., prior to the first injection. Length of the period during which the five injections were made: 1 hr., 5 min.						
69	108	7.6	60.8	563	+	
70	106	9.6	76.8	725	+	
71	100	12.0	96.0	960	+	
72	93	13.0	104.0	1,118	+	
73	89	14.3	114.4	1,285	+	
P. Sodium chlorate (Eimer and Amend, "tested purity"): *16 per cent solution.* Female rats. Food not available for 12 hr., 38 min., prior to the first injection. Length of the period during which the seven injections were made: 1 hr., 12 min.						
74	96	7.7	123.2	1,283	+	
75	90	9.0	144.0	1,600	+	
76	88	10.6	169.6	1,927	+	
77	86	12.1	193.6	2,251	+	
78	86	13.8	220.8	2,567	+	
79	85	14.5	232.0	2,729	−	3
80	85	13.6	217.6	2,560	+	

TABLE 1—*Continued*

Data pertaining to the comparative toxicity of potassium chlorate, sodium chloride, etc.

RAT		DATA PERTAINING TO THE INJECTIONS			RESULTS	
Number	Weight, grams	Volume injected, cc./10	Dose injected. Total, mgm.	Dose injected. Per kilo, mgm.	Lived 2 weeks	Died: min.
*VII. Soaps (castile and sodium oleate)**						
Q. Soap (castile: Eimer and Amend): 4 per cent solution. Male rats. Food not available for 13 hr., 44 min., prior to the first injection. Length of the period during which the two injections were made: 16 min.						
81	182	5.8	23.2	127	−	1
82	178	2.7	10.8	61	+	
R. Soap (castile: Eimer and Amend): 4 per cent solution. Female rats. Food not available for 13 hr., 35 min., prior to the first injection. Length of the period during which the three injections were made: 25 min.						
83	104	2.4	9.6	92	+	
84	103	3.1	12.4	120	+	
85	97	6.8	27.2	280	−	7
S. Soap (castile: Eimer and Amend): 4 per cent solution. Male rats. Food not available for 14 hr., 15 min., prior to the first injection. Length of the period during which the three injections were made: 21 min.						
86	99	5.0	20.0	202	−	1
87	96	3.9	15.6	163	−	2
88	96	2.9	11.6	121	−	2
T. Soap (castile: Eimer and Amend): 4 per cent solution. Female rats. Food not available for 13 hr., 55 min., prior to the first injection. Length of the period during which the two injections were made: 16 min.						
89	91	2.7	10.8	119	+	
90	91	2.7	10.8	119	+	
U. Soap (sodium oleate: Eimer and Amend): 4 per cent solution. Female rat. Food not available for 14 hr., 5 min., prior to the injection.						
91	102	3.1	12.4	122	+	

* The castile soap and sodium oleate used in tests Q to V, inclusive, were the best commercial products available. *The preparations were certainly similar to, and as pure as, the soaps used in dentifrices.* The products dissolved so completely in water (4 per cent) that practically no appreciable mathematical variation was introduced by the filtration of the solutions. In an extension of this phase of the work, purer products will be purchased or prepared, and additional results obtained and published. The influence of the kinds and degrees of alkalinity of the soap solutions employed will also be studied in this relation and the data reported with those for toxicity.

TABLE 1—*Concluded*

Data pertaining to the comparative toxicity of potassium chlorate, sodium chloride, etc.

RAT		DATA PERTAINING TO THE INJECTIONS			RESULTS	
Number	Weight, grams	Volume injected, cc. /10	Dose injected		Lived 2 weeks	Died: min.
			Total, mgm.	Per kilo, mgm.		
*VII. Soaps (castile and sodium oleate)—Continued**						
V. Soap (castile, Eimer and Amend, rats 92–94; sodium oleate, Eimer and Amend, rats 95–101): 4 per cent solution. Sex of the rats: 92–97, female; 98–101, male. Food not available for 12 hr., 21 min., prior to the first injection. Length of the period during which the ten injections were made: 2 hr., 2 min.						
92	92	3.2	12.8	139	−	1
93	92	3.0	12.0	130	−	1
94	90	2.7	10.8	120	+	
95	86	2.8	11.2	130	+	
96	85	3.0	12.0	141	+	
97	83	2.7	10.8	130	−	2
98	106	3.5	14.0	132	+	
99	103	3.6	14.4	140	+	
100	101	3.8	15.2	150	−	1
101	96	3.3	13.2	138	+	

* See the foot-note on the opposite page.

(and convulsions also for the largest doses) occurred after the treatments with *sodium nitrite*, but normal color was gradually restored in a few hours in each of the survivors.

The lethal doses of *soaps*, *potassium chloride*, *potassium sulfocyanate*, and *potassium chlorate*, promptly caused death attended by convulsive movements. After fatal treatment with soap, there was usually a series of convulsive movements followed by cessation of respiration for from fifteen to thirty seconds, or a little longer, then resumption of shallow breathing for a few seconds, then death.

Some of the largest *non-lethal* doses of each of these substances suddenly appeared to be causing death before the calculated volumes had been fully injected, in which instances the injections were then stopped immediately, in the belief that death was imminent; and, of course, only the volumes actually injected are recorded in the table. These striking results, very near the "death line," made it possible

all the more satisfactorily to establish the approximate lethal dose of each of these substances, for albino rats, under the conditions of the tests.

IV. DISCUSSION OF THE RESULTS

The many individual results, recorded in *table 1*, are conveniently reduced to simple terms in *table 2*, which presents a summary of mathematical data for the "minimal lethal dose" and for the "maximum tolerated dose," for each substance, expressed in milligrams per kilo of body-weight of rat, and arranged in the order, downward, of indicated greatest toxicity to indicated least toxicity:

TABLE 2

A summary of data for "minimal lethal doses" and "maximum tolerated doses," in albino rats, for the substances named in table 1

SUBSTANCE TESTED	MINIMAL LETHAL DOSE PER KILO	MAXIMUM TOLERATED DOSE PER KILO
	mgm.	*mgm.*
Sodium nitrite	80	61
Castile soap	121	120
Sodium oleate (soap)	130	141
Potassium chloride	138	132
Potassium sulfocyanate	141	140
Potassium chlorate	258	291
Sodium chloride	2,332	2,243
Sodium chlorate	2,729	2,567

The essential results of the tests described in this paper are shown pointedly by the general data in *table 2*. Injected directly at a standard rate into the venous circulation of albino rats, under uniform nutritive and environmental conditions, potassium chlorate, "*milligram for milligram*," obviously was markedly *less toxic* than such substances of physiological occurrence as sodium nitrite, soaps, potassium chloride or potassium sulfocyanate, but was very much *more toxic* than sodium chloride or sodium chlorate.

The results, considered from special points of view, suggest that much of the toxicity of potassium chlorate is due to its potassium. The data for *potassium* chloride on one side (more toxic), and for *sodium* chlorate on the other (less toxic), strongly support this inference.

These results raise a new question, namely: if *potassium* chlorate is useful in a dentifrice, why would not *sodium* chlorate be more useful, since sodium chlorate is much less poisonous than potassium chlorate (and even less poisonous than an equal weight of sodium chloride), and presumably would exercise the beneficial influence that now is ascribed to potassium chlorate in dentifrices. This question will be raised again, in a later communication.

In the introductory part of this paper it was said that one of the reasons for conducting tests on animals higher in type than those used in Part I of this study was the obvious possibility that the results for toxicity on tadpoles and newts were not representative of relative effects on mammals. We see, however, that instead of divergence there is striking agreement between the general results on rats and the comparable data for tadpoles and newts.

If the comparable data for tadpoles and newts (Part I) are summarized in a manner similar to that shown in *table 2*, the arrangement presented in *table 3* is obtained.

TABLE 3

A summary of data for "minimal lethal doses" and "maximum tolerated doses," in tadpoles and newts, for the substances tested in Part I (1)

SUBSTANCE TESTED	MINIMAL LETHAL DOSE	MAXIMUM TOLERATED DOSE
	per cent	*per cent*
For tadpoles		
Soap	0.02 (2 hours)	
Potassium sulfocyanate	0.10 (51 hours)	
Potassium chlorate	0.10 (14 days)	0.4 (31 days)
Sodium chloride	0.20 (6 days)	1.0 (7 days)
For newts		
Soap	0.02 (20 hours)	
Potassium sulfocyanate	*	
Potassium chlorate	0.40 (10 days)	1.5 (5 days)
Sodium chloride	(above 1.0 for 7 days)	1.0 (7 days: the highest tried)

* Not tested on newts.

The sequence of toxicity shown by the data in the foregoing summary (proceeding from the substance of greatest indicated toxicity to that of the least), is, for *tadpoles:* soap > potassium sulfocyanate >

potassium chlorate > sodium chloride; for *newts:* soap > (potassium sulfocyanate not tested) > potassium chlorate > sodium chloride—the sequence, in each case, for these substances in the summary of the data for rats.

These agreements do not encourage or support the opinion that possibly different relative toxicities would be shown by these substances in men.

The mathematical data have been considered here on a gravimetric basis alone. If toxicity were estimated from calculations of the data on a molecular basis, however, the sequential position of potassium chlorate would be the same; the soaps would exchange places with sodium nitrite as the most poisonous; and sodium chloride would exchange places with sodium chlorate as the least toxic. There would be little significant alteration in the positions of the others in the sequence noted above. After further progress will have been made in this work, all the mathematical data of Part I, and of this part and of the parts to follow, will be compared from both the gravimetric and molecular points of view, and discussed accordingly.

The conspicuous toxicity of soaps, both in Part I and in this part of the study, automatically raises the question whether the introduction of soaps into the mouth, in dentifrices, is as harmless *for the gums* as has been generally believed. Certainly nothing in this work, so far, suggests that the repeated presence of large quantities of soap in the mouth is a matter of hygienic indifference. Special attention will be given to this phase of the subject in a future communication.

The results have an unexpected bearing on the "old sulfocyanate question." It was shown in this laboratory, some years ago (2), that sulfocyanate in the saliva is a waste product and that it has no relation to the incidence or progress of dental caries. The data for toxicity of potassium sulfocyanate seem to support the finding that sulfocyanate is wholly an excretory (waste) product and not in any sense a secretory (useful) substance. Results of special comparative tests with *sodium* sulfocyanate and other sulfocyanates, however, to determine whether or not the toxicity of *potassium* sulfocyanate is due mainly to its *potassium*, or to its sulfocyanate radical, will be required before the later results can be regarded as certainly confirmatory of the earlier. The results of such tests will be published in a future issue of this JOURNAL.

Nitrite as a constituent of saliva is seen to be in a position analogous to that of salivary sulfocyanate. The results of these tests suggest a special study of the origin of nitrite in the body, the quantities of its elimination in saliva, the significance of variations in its salivary and urinary excretions, the possibility of an important natural relation of nitrite to nervous and vasomotor phenomena, and the quantity of nitrite in saliva as an index of such relationships. Such studies will be undertaken.

It was stated, in the summary of results for Part I of this series of studies, that the danger of poisoning in the dentifricial use of potassium chlorate is "not as great as some of the clinical data in the toxicological records suggest," and that potassium chlorate is neither an "*extremely*" dangerous drug nor a "*gravely*" poisonous substance. The results of the tests described in the present paper confirm this conclusion (with emphasis on the words italicized).

The questions whether potassium chlorate, *when taken into the mouth or stomach*, or both, in the form of, say, Pebeco, is absorbed into the blood rapidly enough, and in sufficient proportions, to injure the person, have not been touched directly either in Part I or in the present paper. The experiments comprising Part III, which bears directly on these particular questions, have already been completed and will be described in the succeeding issue of this JOURNAL.

The toxicity of potassium chlorate has been compared in these tests, in the main, with that of comparatively non-toxic or weakly toxic substances. In a subsequent communication will be published the results of a similar study of the toxicity of potassium chlorate compared with that, in albino rats, of such poisons as ordinary salts of mercury, lead, and copper.

Direct response to the question in the title of this paper will follow the presentation of the results of the concluding experiments. It is believed that, by that time, the experimental results will have afforded sufficient evidence to provide a completely truthful answer.

V. GENERAL CONCLUSIONS

In normal albino rats, under uniform dietary and environmental conditions, and injected intravenously, at a standard rate, in aqueous solutions of equipercentage concentration, potassium chlorate was

markedly *less toxic* than sodium nitrite, castile soap, sodium oleate, potassium chloride, or potassium sulfocyanate, but was very much *more toxic* than sodium chloride or sodium chlorate.

The sequence of toxicity for the substances tested, under the conditions stated above, proceeding in order from the substance of greatest toxicity to that of least toxicity, was the following: (a) sodium nitrite, (b) castile soap, (c) sodium oleate = potassium chloride = potassium sulfocyanate, (d) potassium chlorate, (e) sodium chloride, (f) sodium chlorate.

The sequence of toxicity in the results for rats was the same as that (for the substances in both groups) in previous toxicity tests on tadpoles and newts (1).

The relative non-toxicity of sodium chlorate raises the question whether any benefits that may accrue from the dentifricial use of *potassium* chlorate cannot be more advantageously derived from the use, instead, of the much less poisonous *sodium* chlorate.

The results confirm the conclusion previously stated (1), that potassium chlorate is not an "extremely" dangerous drug and that its dentifricial use is not "gravely" perilous, although the findings do not suggest that potassium chlorate may be taken into the body indifferently, or that its use in dentifrices is desirable.

VI. LITERATURE CITED

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(2) KAHN, MAX, and GIES, WM. J. 1912 An inquiry into the possible relation of sulfocyanate to dental caries. *Transactions of the Dental Society of the State of New York*, p. 150 (*Dental Cosmos*, 1913, lv, p. 40).—LIEB, C. C., KAHN, MAX, and GIES, WM. J. 1913. A further study of sulfocyanate in its possible relation to dental caries. *Ibid.*, p. 142 (*Dental Cosmos*, 1914, lvi, p. 173).

(3) TAYLOR, J. M., GREELEY, H., LIMERICK, O. V., MERRITT, A. H., STILLMAN, P. R., HYATT, T. P., BUCKLEY, J. P., and CARNEY, M. F. 1919 Highfalutin dupery: Comment on the falsity of various published claims for certain dentifrices. JOURNAL OF DENTAL RESEARCH, i, p. 503.

CUMULATIVE SUMMARY OF FINDINGS[1]

Mennen's Cream Dentifrice.[2] Not acid in reaction as advertised, but alkaline. Therefore, claims of beneficial action, when used as a dentifrice, as *due to acidity*, are wholly unwarranted. Karshan and Gies: JOURNAL OF DENTAL RESEARCH, 1920, ii, p. 293.

Pebeco Tooth-Paste (and other dentifrices containing potassium chlorate). The opinion recently expressed by Taylor, Greeley, Limerick, Merritt, Stillman, Hyatt, Buckley, and Carney (JOURNAL OF DENTAL RESEARCH, 1919, i, 503), to the effect that potassium chlorate is an "*extremely* dangerous drug" and that "*grave* perils attend the employment" of dentifrices such as Pebeco (which contains potassium chlorate in very large proportion), seems to be an overemphasis on the facts of toxicity. Tests of relative toxicity, on tadpoles and newts by the immersion method and on albino rats by intravenous injection, show that such substances of ordinary physiological occurrence as sodium nitrite, castile soap, sodium oleate, potassium chloride, and potassium sulfocyanate, are markedly more toxic than potassium chlorate, but that the latter is much more poisonous than common salt or sodium chlorate. *Sodium* chlorate, per unit of weight, is less toxic than sodium chloride. The use of the more toxic *potassium* chlorate in preference to the non-toxic *sodium* chlorate, as a constituent of dentifrices, remains to be explained. Gies: JOURNAL OF DENTAL RESEARCH, 1920, ii, pp. 313 and 511.

Pepsodent.[2] Contrary to advertised claims, Pepsodent, used as a dentifrice, *does not* (a) completely remove mucin plaques from teeth, or (b) effectively prevent re-formation of such plaques on teeth, *by a digestive action of contained pepsin "activated" by admixed acid calcium phosphate.* "Pepsodent Bulletin No. 4" presents nothing to support this advertised claim, or to show any error in the denial of the validity of this claim. (1) Gies: *Journal of the American Medical Association*, 1917, lxviii, p. 1278 (reprinted in the JOURNAL OF DENTAL RESEARCH, 1919, i, p. 507); (2) Franke and Gies: *Journal of the Allied Dental Societies*, 1917, xii, p. 360; (3) Franke and Gies: JOURNAL OF DENTAL RESEARCH, 1919, i, p. 511; (4) Gies: *Ibid.*, 1920, ii, p. 299.

[1] A cumulative summary of findings is appended to each of the papers in this series. This plan enables us to present, recurrently, the *sum* of the findings in this series of investigations.

[2] The only findings, known to the authors named at the end of the note, that support the claims referred to, have been published solely in advertisements.

THE ENDOWMENT FUND FOR THE SUPPORT OF THE JOURNAL OF DENTAL RESEARCH

SEVENTH REPORT

WILLIAM J. GIES

Subscriptions to the endowment fund for the support of the JOURNAL OF DENTAL RESEARCH have been received, since the publication of the sixth report (vol. II, p. 325), from the twenty-one additional contributors named below:

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Total amount of the fund	4,074.10
Expenditures from the fund	None
Total amount of the fund invested in United States securities, at rates of interest ranging from 4 per cent to 6 per cent	4,000.00

It is a very great pleasure to call special attention to the generosity of the dental business men and organizations named below, who have contributed effectively to the permanent support of this JOURNAL without qualification, condition or reservation, and who have shown, by the cordiality of their specific approval, that they wish to encourage and foster the development of independent research and scientific journalism in dentistry.

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Contributions to this permanent endowment fund, *in any amounts*, are invited from *all who may be interested in the welfare and continuing success of this* JOURNAL.

SCIENTIFIC PROCEEDINGS OF DENTAL AND STOMATOLOGICAL SOCIETIES

THIRD SECTION

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A LIST OF THE NAMES OF THE ORGANIZATIONS THAT HAVE VOTED TO MAKE THE JOURNAL OF DENTAL RESEARCH THE MEDIUM FOR THE OFFICIAL PUBLICATION OF THEIR SCIENTIFIC PROCEEDINGS

ASSOCIATION OF THE ALLIED DENTAL SOCIETIES, INC.

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HARVARD ODONTOLOGICAL SOCIETY

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METROPOLITAN DISTRICT OF THE MASSACHUSETTS DENTAL SOCIETY

HARRIET NEWELL LOWELL SOCIETY FOR DENTAL RESEARCH, OF THE HARVARD UNIVERSITY DENTAL SCHOOL

COLUMBIA UNIVERSITY BIOCHEMICAL ASSOCIATION

In the case of the last named organization, only the dental and stomatological parts of its proceedings will be published in this JOURNAL.

A standing general invitation is extended to dental and stomatological societies, everywhere, to add their names to the above register.

PROCEEDINGS OF THE FIRST DISTRICT DENTAL SOCIETY OF THE STATE OF NEW YORK

WILLIAM B. DUNNING, *Editor*

Meeting held February 2, 1920. Academy of Medicine, New York City. President Frederick C. Kemple, in the chair.

Scientific proceedings. *Paper* by Clarence J. Grieves, D.D.S., Baltimore, Md., on a "*classification of teeth the diseased pulps and apices of which are related to infective focal and systemic sequelae*" (page 327). *Discussion* by Drs. William B. Dunning, Charles F. Ash, N. T. Shields and M. L. Rhein, of New York City; and Dr. Grieves (this page).

DISCUSSION OF DR. CLARENCE J. GRIEVES' PAPER ON A "CLASSIFICATION OF TEETH THE DISEASED PULPS AND APICES OF WHICH ARE RELATED TO INFECTIVE FOCAL AND SYSTEMIC SEQUELAE" (PAGE 327)

BY DRS. WILLIAM B. DUNNING, CHARLES F. ASH, N. T. SHIELDS AND M. L. RHEIN, OF NEW YORK CITY; AND DR. GRIEVES

Dr. W. B. Dunning (New York City): Dr. Grieves has very considerately summarized his admirable paper under ten conclusions. I think I can best serve my purpose as a discusser by going over those conclusions, and touching lightly on them. I can only judge as a practitioner, not as a bacteriologist or laboratory man; and I take it most of my audience approach the subject from the viewpoint of the practitioner, and not as scientific specialists.

Under his *first conclusion*, Dr. Grieves says: "Dental caries is dual in character, being a chronic tooth-wasting disease, with tissue necrosis or liquefaction superficially, and pathogenic forms irregularly invading the hard underlying dentinal matrix. This sepsis begins in the granular layer of Tomes, and is the primary infective focal portal. Therefore, because dentin is hard or free from soft caries, it is not necessarily free from bacteria; and some effort must be made to sterilize every cavity-preparation in vital teeth." Of this I heartily approve.

Going to *Conclusion 2:* "The normal pulps of vital teeth should be devitalized rarely; and only when devitalization is important for the construction of such occlusal restorations as are impossible by other methods and absolutely necessary to general nutrition; recalling the 70 per cent of occlusal efficiency of bridges, as compared with the 30 per cent of dentures. . . . . "

After all, is not the moral of this paper the fact that we must conserve the pulps of teeth in a state of health? Must we not make our best efforts in that direction, since, when we go into pulp removal, we step into hopeless quicksands? It is a subject that is so vast and shifting at the present time, that the practitioner feels the one definite thing to grasp is the chance of saving a pulp in a living state.

Dr. Grieves speaks of the greater susceptibility of youthful pulps. That is an interesting point. I wish Dr. Grieves would develop that point further in his discussion; because it has seemed to me that, in my efforts to conserve the pulp of, let us say, a first permanent molar in a child, it has been attended usually by success; and I attributed the outcome largely to the rich vascularity of that tooth, and the abounding vitality of youth. It seemed to me there was a definite connection there, and that the possibility of conserving vitality in a youthful tooth was greater—as we know, it is of greater moment to the patient, since the apex of the root is not fully developed.

"*Conclusion 3.*—Any pulpless tooth, the periodontal membrane of which is gingivally purulent, should be removed, no matter how perfectly filled, or how well repaired subapically, whether periapically diseased or not; for, at any time, it might become diseased by the gingival pathway. If pulp devitalization is absolutely necessary, it should be practised on only such pyorrheal teeth as are not swaying to the middle third; or those cured (?) by periodontic treatment; and then, on only those in which apical hyperplasia has closed in part the foramina as noted. (Such hyperplasia is usually demonstrable, clinically, and by good radiographs.)"

I should stand as a "mild reservationist" on that point. If the apical condition is good, careful scaling and polishing will save many such teeth. The effort is often worth making.

"*Conclusion 4.*—Unless pulp symptoms or exposure arise in eroded, abraded or senile teeth, it is meddlesome practice to risk pulp devitalization and defective canal filling, just because pulps *might* degenerate, the exception to this rule being pyorrheal teeth, which might be infected by the gingival pathway. . . . . "

That, I think, is a splendid conclusion. It *is* meddlesome practice, and there is no better term for it.

"*Conclusion 5.*—Cases in Class IV (fresh surgical exposure of symptomless pulps, made through hard dentin, even at the risk of its being infected) are hopeful. Capping should be attempted, with frequent clinical and radiographic survey; but the slightest periodontal symptoms demand immediate pulp removal. This is especially true of young unfinished apices. . . . . "

I think most of us will agree to the sound wisdom of that conclusion.

"*Conclusion 6* (for the teeth of Classes I, II, III, IV and V, diagnosed carefully and promptly).—If pulps can be removed under as nearly aseptic conditions as possible, by careful chemico-mechanical instrumentation, leaving only inaccessible vessels and vital pulp-shreds in the immediate foraminal openings of a vital apex, which has not been cauterized nor over-sterilized; or, if these strong agents can be used without their passing out into the membrane (though conservative operators prefer milder medicaments)—if all of this can be accomplished without perforation and encapsulation, avoiding infection from débris, then, any canal-filling method, as nearly aseptic as can be, that will close these openings well into, but not through the apex, will insure apices infected as little, and filled as much, as possible and a functional tooth."

I can simply say in passing, that it has been my practice for a number of years to reach the apex of the tooth, if it were possible to do so. I do think we should be careful

not to carry strong chemical agents through the apex—to cease our operation the moment we are aware of our arrival at that point. Consider what the slightest burn of your hand would be. Let us say a heated pin point reaches your hand. Instantly it is removed. A slight cauterization has occurred, and nature takes care of that. I think to reach the apex of a root without carelessly carrying it through the apex, is correct practice. I prefer to do that, rather than risk later the death of the organic shreds that lie near that apex.

It seems to me the point made by Dr. Grieves, that the supplemental foramina are rarely explored, is a little overdrawn. The beneficial effect of the sodium-potassium paste is that it will work into these supplemental foramina, whether there are two or twenty. That is frequently proved to our great gratification, after taking a picture following the root operation, when we observe those multiple delicate foramina actually filled with gutta percha, each running at such an angle from the pulp canal that it would be mechanically impossible for any broach to follow. In such cases, I have emptied these delicate canals of their contents, and they have become filled. I have not been there with an instrument; but it has been through the use of this chemical agent that I have been able to do that—and not by any mechanical means. You have all had that experience.

As for encapsulation: I have encapsulated many root apices, and I have generally found that where it has been performed carefully and with proper aseptic precautions, you do not hear much more from such teeth. I realize that that is rather an empirical way to speak of it; but, are we not all practising more or less empirically? We are indebted to Dr. Grieves for the great amount of work he has put into this subject; and yet he is full of reservations, and so is every one else. Our actual knowledge is very meagre. We are endeavoring by all means to increase our knowledge, but we must ever lean on the staff of experience. Therefore, speaking from a practical point of view, I am inclined to reach the ultimate apex of the root, and fill that, preferring to obtain a slight encapsulation, if possible.

"*Conclusion 7.*—If pulp removal and canal filling can be accomplished according to the conditions specified in *Conclusion 6;* if the lamina dura is definite, and surrounding bone cancellations appear to be normal, as in adjacent vital teeth; if there are no clinical symptoms and the apex is not hypoplastic, but contains a filling well into, but not through it, with no visible open canal beyond; then, a pulpless tooth so situated should be retained, as it is functional and harmless."

I think so, too; but I like to see it completely filled.

"*Conclusion 8.*—General nutrition may be seriously disturbed, and great distress caused the patient by indiscriminate removal of Class-V (apically well filled) teeth, for diagnostic purposes or because infection is suspected. Such operations are frequently peremptorily ordered by medical men in their anxiety to cure disease and in ignorance of oral pathology. Obviously, such teeth are extracted by a dentist, who, if he does not protest or refuse, but operates following a fad or through fear of, or to please, his medical confrère, undoubtedly damages the patient's health and lowers the dignity of his profession."

The subject of indiscriminate extraction is properly being very seriously considered at the present time. I think we have all been a trifle reckless, perhaps; and yet, on the other hand, surgery must ever be our surest aid in many cases.

"*Conclusion 9.*—With the exceptions mentioned, all periapically diseased teeth, when clinically and radiographically definite (Class VI), should be promptly extracted and

sockets curetted. If there be the slightest question, it is best to err radically, and remove a doubtful tooth, than to risk inevitable damage to adjacent teeth, attaching tissues, or the maxillae and possibly the patient's health."

A great many such teeth have not been extracted in the past, and I personally have had the temerity to undertake the treatment of such teeth; to be sure, not extremely diseased ones, but some that were definitely diseased apically—where I have carried on medication of a mild character for a long time, where I have obtained encapsulation such as Dr. Rhein recommends, where apparently bone regeneration or something resembling it has occurred. Whether there will be an after-clap, or a breaking down in years to come, remains to be seen.

As to *Conclusion 10*, I should like to ask Dr. Grieves to give us further information on the point he makes, as follows: "Dentists generally must surgically remove all such lesions, regardless of whether they be infected or not; because a real chronic bone pathology exists about the tooth and its cell reactions often *simulate* malignancy—it eventually proliferates, undermining the denture. So, obviously, the dentist, quite as urgently as the physician, and certainly the patient, desire prompt action to prevent further bodily invasion." Does it "simulate" or "stimulate;" or does Dr. Grieves mean both?

Under Class VI, I should think further knowledge might perhaps enable us to determine which of the badly diseased teeth must be removed, and those which may, with a certain degree of safety be carried through treatment, and perhaps filled. I refer to a bacteriological examination of the contents of such canals by sealing, for instance, in a canal for twenty-four hours, a paper point or cotton fibers saturated with sterile broth; that being removed the next day and a smear made. The nature of the invading organism has so much to do with the prognosis of the case that, as our knowledge increases, we should be able quickly to determine the hopeful cases in this way.

But after all, does not this whole subject hark back to preventive dentistry? The most inspiring work we can do is for the child. There has been a great forward urge in the direction of oral hygiene in recent years, but I often think we are remiss in not following up, with sufficient exactness, the children who come under our care. In looking back over my own modest attempts at saving teeth, I find the greatest satisfaction in those little fillings in developmental grooves in the newly erupted teeth that have been placed while the cavity was just at the point of inception; when it was, so to speak, a "pin-head" cavity. Such a cavity is very minute—scarcely at first admitting the exploring instrument—and when it is carefully filled, that little incident in the child's tooth is closed for the rest of his life, in many instances. We all know with what appalling speed a slight developmental fault in a young tooth will become a serious decay. Is it not a disgrace for any practitioner to find it is necessary to make a compound gold inlay in a first molar, for instance, for a boy of fifteen, supposing of course that that boy has been under his care since childhood? Should not that have been prevented by proper care, so far as the practitioner was concerned? We should maintain an aggressive attitude toward our young patients and their parents, insisting upon examinations at least every three months, to treat this incipient decay, and to train the individual in the personal care of the teeth.

We are all interested in dental nomenclature, and Dr. Grieves' subject tonight has involved the use of a pretty wide vocabulary; but it seems to me we have not yet agreed upon the happiest word to express a tooth that is no longer vital. Dr. Grieves has used, among others, the word "devital." The Latin "de" means "away from." But

is not "non-vital" a better term? It means the vitality is not there, is non-existent. I believe we should say we *devitalize* ("take away" the vitality of) a tooth, but having done so, we have a *non-vital* tooth. This seems to express the condition more accurately than other terms.

Dr. Charles F. Ash (New York City): We are greatly to be congratulated on having heard Dr. Grieves read his masterly discourse. I am not competent to discuss all phases of this paper—in fact, very few. When Dr. Grieves referred to the proportion of teeth which were found to be healthy, and those which were found to be unhealthy, in their apical areas—among those which had been filled—I wanted to make this suggestion; that we bear in mind the fact that the majority of these teeth are teeth which were treated under the rather imperfect methods of sterilization and surgical asepsis that prevailed to within a few years ago. I wish to emphasize the possibility of a hopeful sign in the increase of favorable ultimate results, in the filling of root canals, through the spread of information in regard to proper surgical asepsis in the handling of such teeth when we are treating and filling the roots. I believe that, if we could take a census of the teeth that are treated by the intelligent majority of dentists who are endeavoring to fill roots under surgical aseptic conditions, for the next five years and examine them ten years later, the proportion of good results would be much larger than 52 per cent. I think we all recognize that, in spite of the care taken by practitioners in the past, in their root-canal work, they were not aware of the proper aseptic surgical procedure. Therefore, with our present knowledge and improved technique, we may reasonably hope for a very much larger proportion of favorable results in the future.

Dr. N. T. Shields (New York City): I was very much surprised tonight to hear Dr. Grieves report that there were but fifty-two good root fillings out of every hundred. In the magazines which I read, which are quite numerous, I see very startling statements. One gentleman, whose name I will not repeat (I am ashamed of him) said that if any man could produce a single case where a pulpless tooth had lasted six months without bad results, there would be nothing to this argument.

I believe this is as sincere as any caution I can utter: When you cease to force the root filling through the apex, you will get your greatest rewards. Dr. Grieves says he does not know why, but that his best result has been obtained where the filling has failed to go through the apex.

Dr. M. L. Rhein (New York City): It is impossible for me to decide, at this time, how much I agree and how much I disagree with Dr. Grieves' classification. The subject of classification of this general subject is not a new one. European writers have given a great deal of attention to it. Some very valuable work in this respect was done by our late enemies before the war, especially in Budapest. It is a very difficult matter to produce a proper classification, and my reason for not having gone into the subject is the fact that a number of perplexing bacteriologic questions are involved.

I realize that it is impossible for me, in a few minutes, to discuss the general conclusions with which the essayist has favored us this evening, but I want to emphasize very strongly what Dr. Ash has said in regard to one fact: the statistics Dr. Grieves has presented, in regard to pulp therapy, are not worth consideration as statistics. This statement is not a reflection on the essayist—it is simply a reflection on the character of the root therapy from which these statistics were drawn. This situation has been

alluded to by Dr. Ash. I shall not take up your time by going into details regarding the antiquity of the class of root therapy from which most of these statistics have been taken.

The question of root-canal therapy is one that embraces a very particular point, and that is, that there is no operation that I know of, as a medical man, outside of brain surgery (and I would even include a large amount of brain surgery) that requires the same amount of skill, patience and time. The most important requirement of all is time-consuming attention. Given sufficient time and attention, the operation is one that will do the things the essayist says cannot be accomplished.

The essayist has spoken rather dogmatically, and I reply somewhat dogmatically, because the proof of the pudding is in the eating thereof. When I show hundreds of roentgenograms—some of them dating back to work done almost thirty years ago—showing absolutely, so far as a roentgenogram can show, normal tissue with thorough encapsulation of gutta percha on the apices of the roots—I definitely refute the essential point of criticism in the essayist's paper.

One of the strong points the essayist has made is his argument against the use of powerful medicaments in the periapical region. There is a lot of truth in that statement, but it does not cover the kind of technique that we are teaching in the First District Dental Society at the present time. If you use a therapeutic agent, such as sulfuric acid, or any of the other inorganic acids, and pass some of it through the foramina, and attempt to destroy the diseased tissue in this way, you leave in that tissue a permanent residue of the medicament you use. We use practically very little of the pharmacopeia in our technique. We use that wonderful combination of sodium and potassium to which Dr. Grieves objects; but how does this act? It destroys what it does destroy in the same manner that the electric cautery would destroy it. The destruction is produced by intense heat. It is the oxidation resulting from the reaction between the water in the tissue with the sodium and potassium that produces this high heat, and that burns out the tissue in the same way that the surgeon would do it, if he could cut down there and remove it. There is no difference between the way sodium-potassium destroys tissue in the periapical region, and the way the surgeon would destroy it, because when you are through with the treatment no residue is left to interfere with regeneration. I concur in the essayist's objection to the use of a medicament which in any way interferes with regeneration. Sodium-potassium does not do it. Of course, its use can be abused. Used rationally, however, it is one of the greatest aids to the radical cure of an apical region with a granuloma. I say "cure" because we have cured them in this way, following it up with electrolytic medication, and because, for many years afterward, the roentgenograms show normal tissue—a regeneration!

I do not know on what basis, or by what authority, Dr. Grieves says there can be no alveolar regeneration. It is very difficult to prove the occurrence of alveolar regeneration, because the alveolar walls are extremely thin. To disprove it is also just as difficult, and it becomes more or less a matter of dogmatic statement. According to a general law in physiology, lost tissue will restore itself under ordinary circumstances, especially in the absence of infection.

In regard to the character of the teeth that the essayist speaks of, in which no evil results occur, let me say that, if all the imperfectly filled root canals were productive of systemic disease, such as we obtain in a certain percentage of cases, three-quarters of the civilized world would have been destroyed by the activities of the dentists of the past. Fortunately, so far as my observations have gone, this question of severe systemic dis-

eases comes down practically to the character of the attacking organism. I have statistics from about 900 bacteriologic examinations. It is a very remarkable fact that of those cases, all that are associated with severe metastasis show one thing, without exception, and that is, infection by Streptococcus viridans—not the ordinary hemolytic streptococcus. I have many cases of streptococcus infection—just the ordinary hemolytic form—in which there is no evidence of metastasis. I am not ready to make a report on this subject, but I have had so many such cases that clinically I have a very strong conviction on this point. Absolute sterilization of the periapical region is not necessary. Absolute artificial sterilization of any part of the body is practically an impossibility. This has been very clearly shown in the wonderful war surgery of Carrel and Dakin, where their best results were obtained under conditions that did not involve absolute sterilization.

The question of obtaining an absolutely sterile periapical region, therefore, means comparatively very little; too much importance has been placed on that particular point.

I want to emphasize what Dr. Dunning said about those by-paths of the pulp that go off into little outside canals, and that the essayist spoke of as impossible of handling in a thorough manner. We do not look at it in that way. We have our failures, to be sure; but, when we have a canal, or any line leading from it, in which every particle of pulp tissue cannot be removed, we feel that such a tooth should be extracted. I could point to, and I have the roentgenographic records of, many cases such as the essayist says fulfill the requirements in Class V, where you get a healthy periapical region though the filling does not go through the foramen. I have records of such work, done by myself; in some cases periapical infection resulted later. I have the roentgenographic records of infection following every form of root-canal therapy that I know of, but I have yet to see a tooth with a perfect encapsulation (and I think this Society knows by this time what I mean by that) where there has been re-infection. Where I have found re-infection in my own cases and have had to extract the teeth, I have also found invariably extra foramina at some point that had not been encapsulated, and which were the cause of the recurrence of infection.

I take serious exception to the essayist's statement as to the impossibility of absolute sterilization of the dentine. Dr. Dunning alluded to this. The bacteriological work we have done hundreds of times shows attainment of absolute sterility in those conditions.

I want to assure Dr. Grieves that I have greatly enjoyed his paper. I believe he is in a progressive state of mind in regard to this subject. I want to assure him that we can cure certain forms of diseased conditions in the periapical region, we can eradicate certain pathologic conditions, and we can favor regeneration of the alveolar structure; and, if we can get a perfect encapsulation, we can leave the ends of the roots in the condition the essayist requires—impervious to infection. We can do that mechanically with gutta percha.

One word more in closing. The essayist speaks of the contraction of gutta percha. I don't know what kind of gutta percha he has in mind but there is no effect on base-plate gutta percha at a temperature of 100° F. It is unaffected by this temperature. The base-plate gutta percha we use, dissolved in chloroform, returns, when we are through with it, to its initial condition. There is neither contraction nor expansion. If there were contraction, the x-ray would demonstrate it, but the x-ray fails to show it.

Dr. Grieves: I have not much to say, except to thank you and answer a few questions, and have the annual quarrel with my friend, Dr. Rhein.

I want to reply to Dr. Dunning regarding the conditions that occur in children's teeth, where pulpitis is of the chronic hyperplastic type—the common pulpal tumor with many granulations. I did not say the teeth die. On the contrary, I said they were very hopeful and might resist apical infection a long time. The apex, I believe, is protected by a granulating line of demarcation: many slides prove that. That frequently saves us, and should encourage capping the symptomless pulps of children's teeth. But I warned against the collateral infection that might pass through the dentine, because of low resistance, and probably reach the pulp.

The Chicago report I used was prepared from thousands of pictures by Drs. Callahan, Grove, and Hyatt, and by myself. It was very discouraging. Dr. Black's report went over the same ground with better results. My conclusion was that if 52 per cent were saved from apical disease, with all the bad work then done, how much more hopeful is the outlook with the present technique.

Of course, Rhein radiographed teeth thirty years ago and knew, then, all about accessory foramina and how far fillings went to or beyond apices; so that is hardly worth discussing.

Supplemental foramina are a matter of anatomy and not personal opinion, and 40 per cent is a very low estimate of the proportion of their occurrence. From four to twelve accessory foramina are very common. I have noted the effect of contracting gutta percha dissolved in chloroform, which is not the same as gutta percha points pumped into chloroform-rosin that prevents contraction by adhering to the apex, as Callahan said, regardless of statements to the contrary. I have seen chloro-percha pumped into one or two accessory, but never into all, the foramina; so they are rarely all filled.

I have worked with many medical men who condemn apically well filled pulpless teeth (Class V). I had thus the opportunity to check up, in pathology and bacteriology, by opening into alveoli (as for apicoectomy) over teeth which I personally would not have condemned. I have not seen an apex, where the periodontal membrane was denuded and that could be encapsulated, that was not diseased. I have fished out many encapsulations with a bacteriologist standing by to check up my procedure; and we not only found infection but pathology, and occasionally hemolyzing streptococci.

When periapical pathology and infection occur, whether encapsulated or not, if the periodontal membrane is not attached at all points to the apex, I consider that tooth diseased. I do not know how to cure it, so I am safer in extracting that tooth. In reverse, if there were no evidence of loss of attaching tissues, I should retain any apically well filled pulpless tooth.

The principal reason for that conclusion is, regardless of whether there is infection or not, that every dentist should want to remove a real bone pathology from every tooth. Suppose it is sterile: it is a mass of granulations; it did not exist there before the apex was involved and does not exist on adjacent teeth. If you go down into it, you will probably find some part infected. Granulation tissue has no more business in the jaws than in any other bone in the body. When it persists, it takes the turn of an "oma," and grows, and undermines that particular tooth and its neighbors. The fact that they occasionally compensate in the middle-third periodontium, and get so firm you almost have to "blast" them out, does not matter. You have a bone pathology there, which will surely get larger at any period of lowered resistance. It seems to me to be common sense for the dentist and the physician to agree that such teeth and all diseased tissue should be taken out.

Dr. Dunning: Does it also stimulate malignancy?

Dr. Grieves (*in conclusion*)): I said, "*simulates*," not "stimulates," malignancy. I have compared many slides of granulating areas and can show some cases with the epithelial spurs. I know they proliferate. I would not for a moment say that they stimulate malignancy, but I do believe they *simulate* it.

Regarding the action of sodium-potassium: I had the good fortune to be with Dr. Callahan when he pushed it into fine broach holes made in fresh bone and later made and examined sections. Anyone who saw those slides, saw what it does to bone. When sodium-potassium acts on bone cancellations, it is quite true it takes out everything but it does not bring in anything. In bone surgery, you have a hemorrhage, and it is the new blood-cells with bone-forming elements that make repair. There is no normal hemorrhage after the burning of bone with sodium-potassium.

I thank you very much for your close attention to such a tedious paper. In talking to patients or medical men, I would suggest that you do not speak of metastases. There have been very few cases in our experience that were metastatic. They were generally toxic. It is very hard to establish the fact that there is an actual escape of microörganisms into the blood stream. The cases of viridans poisoning, where septicemia actually occurs, are always fatal and I am glad to say are rare. I believe the viridans matter is very much over-done. The safer ground, until we know more, is to assume that systemic results occur as toxemias and not as metastases.